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THE ETIOLOGY OF YELLOW FEVER

AN ADDENDUM.

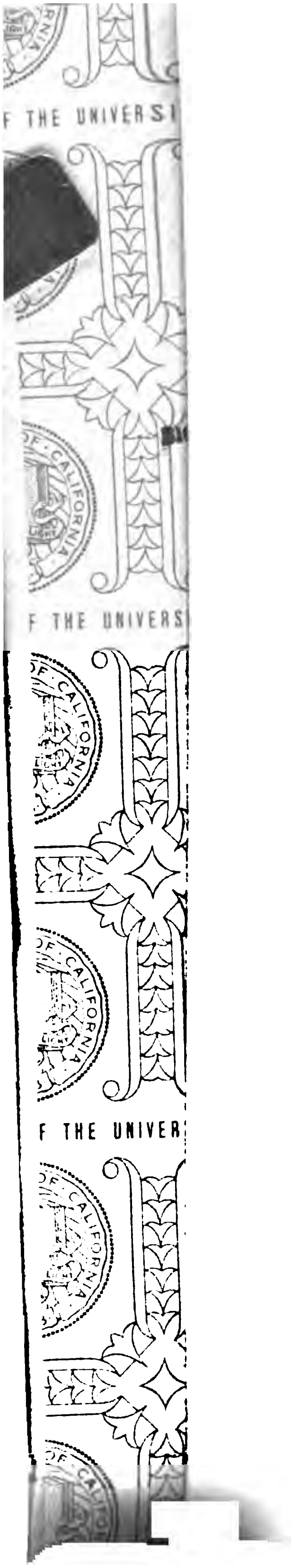
Read at the Thirty-first Annual Meeting of the American Public Health Association, held at Washington D. C., October 26-30, 1903.

JAMES CARROLL, M.D.
First Lieutenant and Assistant Surgeon, U. S. Army.
WASHINGTON

Reprinted from The Journal of the American Medical Association, November 28, 1903.

CHICAGO:
PRESS OF AMERICAN MEDICAL ASSOCIATION
ONE HUNDRED AND THREE DEARBORN AVENUE
1903.

Parasitic Protozo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THE ETIOLOGY OF YELLOW FEVER.

AN ADDENDUM.

JAMES CARROLL, M.D.
First Lieutenant and Assistant Surgeon, U. S. Army.
WASHINGTON.

In a paper entitled "The Etiology of Yellow Fever, a Supplemental Note,"[1] the Army Yellow Fever Board (which was then composed of the late Major Walter Reed, surgeon, U. S. Army, and the writer) stated that no results worthy of record had been obtained from the examination of infected mosquitoes, either when dissected in the fresh state or when studied in serial sections after being hardened and embedded. They also reported that two out of three non-immunes had been infected with yellow fever by the subcutaneous injection of 1.5 c.c. of blood serum drawn from a yellow fever patient and subsequently passed through a new Berkefeld filter which was afterward found to be impervious to the *Micrococcus aureus.*

In the latter part of July of the present year there appeared from the Government Printing Office the Report[2] of Working Party No. 1 of the Yellow Fever Institute of the U. S. Public Health and Marine-Hospital Service. These observers (after repeating some of our experiments) presented additional confirmatory evidence in support of the mosquito theory of the transmission of yellow fever and in that respect as well as in the negative results of their blood examinations their conclusions agreed with those of the Army commission.

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[1] Read before the third annual meeting of the Society of American Bacteriologists, Chicago, Dec. 31, 1901, and published in Amer. Med., Feb. 22, 1902.

[2] Report of Working Party No. 1, Yellow Fever Institute.—"A Study of the Etiology of Yellow Fever," by Herman B. Parker, Assistant Surgeon; George E. Beyer, Acting-Asst. Surgeon; O. L. Pothier, Act.-Asst. Surg., March, 1903, Washington, Government Printing Office.

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They reported negative results, however, from two (subcutaneous?) inoculations with fresh yellow fever serum after it had been passed through a Berkefeld filter. The quantity of filtered serum injected in these experiments was in one instance one forty-fifth and in the other one-fifth of that used in our own cases.[3]

The most interesting and striking feature of the report was the description of a protozoan parasite to which the name *Myxococcidium stegomyiæ* has been given. This parasite was found with regularity[4] in the appropriate mosquitoes that had bitten patients affected with yellow fever, these mosquitoes after varying periods being killed, sectioned and stained. Simple staining with Bismarck brown or hematoxylin were the methods used by these observers for the demonstration of the parasite. Five forms are described: (*a*) fusiform bodies found in the stomach and ventral diverticulum;[5] (*b*) fragmentation bodies lying in an albuminoid mass within the same diverticulum;[6] (*c*) compact aggregations of spores (called oöcysts) occurring in spherical masses[7] in similar albuminoid material in the same location; (*d*) sporoblasts (?) appearing as single or aggregated spherical or ovoid cells in the albuminous material in the diverticulum and in the tissues of the thorax;[8] (*e*) spores (?) containing sporozoites (?) in the salivary glands.[9] One or more of these stages were said to have been found in a large proportion of the infected mosquitoes that were killed not earlier than the third day after being fed on the blood of patients during the second, third and fourth days of the disease. The cycle of development of these so-called animal parasites as given by the authors is about as follows: On the third or fourth day, after the disappearance of the blood, the stomach was found to contain varying numbers of small fusiform-shaped protozoa arranged singly or in groups and containing a central, oval nucleus. Certain of these forms (budding forms) were interpreted as indicating a terminal conjugation; after conjugation the resulting zygotes migrated through the stomach wall into the diverticulum and underwent further development in a mass of albuminoid material in the latter sac. Here were found also the fragmentation bodies and oöcysts (?), the latter represented by rather compact spherical aggregations of round or ovoid bodies, the sporoblasts (?). After their liberation these sporoblasts wandered into the thoracic end of the diverticulum, passed through its wall and the tissues of the thorax until the salivary gland was reached. After penetration of the gland sporozoites were developed and liberated.

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[3] Reed and Carroll: Amer. Med., Feb. 22, 1902.

[4] Report of Working Party No. 1, Conclusion 8, p. 8.

[5] Figure 15 of the report.

[6] Figure 16 of the report.

[7] Figures 17 to 24 of the report.

[8] Figures 25 to 28 of the report.

[9] Figures 29 and 30 of the report.

Availing myself of the announcement that type specimens were preserved in the collection of the Hygienic Laboratory of the U. S. Public Health and Marine-Hospital Service, I requested permission to examine them, and I desire to acknowledge here the great courtesy and kindness extended to me on two such occasions by Dr. Herman B. Parker, the chairman of the working party. On subsequently re-examining our own sections of infected and non-infected mosquitoes which had already been studied more than two years before in conjunction with my honored colleague, the late Major Walter Reed, surgeon, U. S. Army, and Dr. William M. Gray,[10] microscopist to the Army Medical Museum. I was unable to satisfy myself that a single one of our infected mosquitoes contained any of the fusiform, round or oval bodies described in the report of the

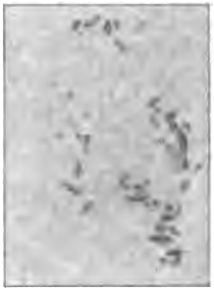

Fig. 1.—This is Figure 15 from the Report of Working Party No. 1, etc., and shows the fusiform bodies found in the stomach and diverticulum of infected mosquitoes.

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[10] Practically all of our insects were embedded, cut and mounted by Dr. Gray.

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working party of the Yellow Fever Institute, but forms similar in appearance to those shown in Figure 16 of the report were occasionally found in the stomach contents. From their striking inequality in size and the absence of any structural details in these "fragmentation" forms, I regarded them even in the type specimens above referred to as masses of amorphous granular material. The bodies containing sporozoites[11] (?) which were found in the salivary gland have been abandoned by these investigators because they no longer regard them as of importance.[12] It is noted that in a more recent article by the chairman of the working party all mention of these supposed sporozoites is omitted.[13]

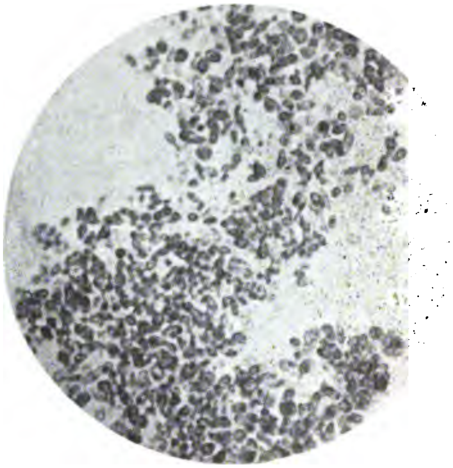

Fig. 2.—Photomicrograph, showing yeast cells in the diverticulum of a non-infected banana-fed insect from Havana. Spherical cells predominate, but fusiform and budding (conjugating?) cells are also shown. x600.

The report does not state in what relative number of insects each of these forms was seen, but I learned from Dr. Parker, on inquiry, that the forms interpreted by them as fusiform protozoa were the only ones found to be constantly present. These are described as 3 to 4 microns long by 1.5 to 2 microns broad, and containing a nucleus.[14] Appearances interpreted as evidence of conjugation are seen in certain of these forms where two of the ovoid bodies, varying more or less in size, are joined by a slender protoplasmic constriction[15] (see Fig. 1); others show a minute bud projecting from one or both poles. The nucleus appears as a small brightly refractive unstained spot when the bodies are stained with hematoxylin or Bismarck brown, either of which colors the whole cell. The bodies that impressed me as being of the greatest importance, if constantly present, were the oöcysts (?) in the albuminoid mass[16] and the sporoblasts[17] (?) in the connective tissues of the thorax.

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[11] Figures 29 and 30 of the report.

[12] Dr. Herman B. Parker, chairman of the party, is my authority for this statement.

[13] "The Etiology of Yellow Fever," by Herman B. Parker, M.D., Jour. of the Assn. of Mil. Surg., vol. xiii, No. 4, Oct., 1903.

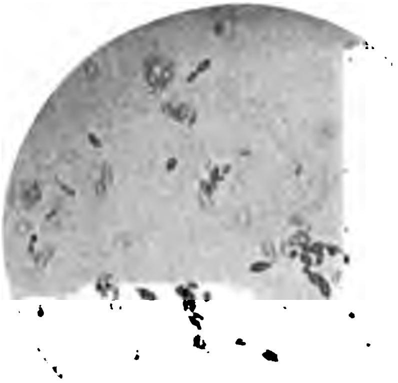

Fig. 3.—Photomicrograph, showing fusiform and budding cells in the diverticulum of another non-infected banana-fed insect from Havana. x600. These mosquitoes were sectioned and examined early in 1901.

With these pictures fresh in mind, on re-examining our slides prepared from infected mosquitoes, I failed to find any of the forms described, excepting, of course, the "fragmentation" bodies[18] to which I could attach no significance. On re-examining sections of normal (non-infected) mosquitoes the results were different, for in one of these, cut in cross section, the ventral

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[14] Report of Working Party No. 1, etc., p. 31.
[15] Loc. cit., Figure 15.
[16] Figure 23 of the report.
[17] Figures 27 and 28 of the report.
[18] Figure 16 of the report.

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diverticulum showed large numbers of spherical and fusiform cells, some of them corresponding in size and form to those described by the working party, but stained brilliantly with eosin (see Fig 2). The majority however, were spherical and a few were elongated into large rod forms resembling detached hyphæ. The smallest contained a single refractive spore, of minute size. The larger fusiform or flask-shaped bodies often contained several spores and in a few instances a single spore was seen to have passed into a projecting bud of protoplasm. A few hyphal filaments about 2 to 3 microns broad and 15 to 20 microns long exhibited a central row of spores arranged with a fair degree of regularity. The characters described justified the conclusion that these cells were blastomycetes that had gained access to the body cavities of the insect with food or water.

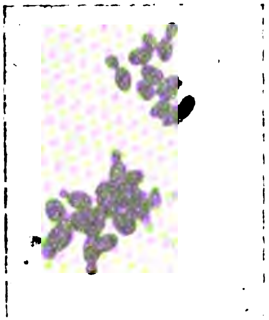

Fig. 4.—Photomicrograph, showing the ellipsoid yeast obtained from banana. Deep colonies in an agar culture two days old. Stained by Gram's method. x600.

In another non-infected insect cut in longitudinal section, fusiform, ovoid and flask-shaped cells (see Fig 3) corresponding in every way with the fusiform stage of the *Myxococcidium stegomyiæ* as seen in their type specimens, were found throughout the ventral diverticulum, but were present in the upper portion of the stomach only and away from the blood mass. This insect had been permitted to take a meal of normal blood some hours before it died, and it is of interest to note that the organisms were found not within or near the residual mass of ingested blood, but only in the upper third of the stomach and associated with large numbers of bacteria. Their location suggests that instead of migrating through the wall of the stomach to the diverticulum[19] they were carried over mechanically from the diverticulum into the stomach.[20] The large majority of these cells showed a single central refractive vacuole (not spores in this instance) of minute size; a few contained two of them. Hyphal forms were not observed. The sections were stained originally with thionin and eosin on a number of slides and after decolorization the cells described were found also to stain perfectly with hematoxylin, Bismarck brown (these two were the methods used by Parker, Beyer and Pothier), and by Gram's method. Similar results were obtained

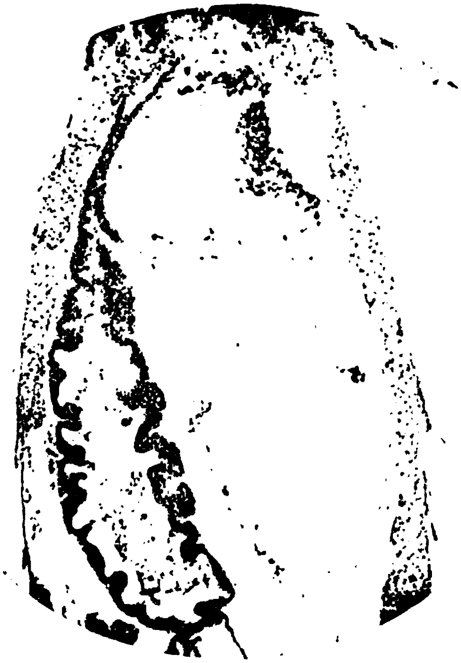

Fig. 5.—Photomicrograph, showing masses of yeast cells in the stomach and diverticulum of an insect fed on yeast culture and banana. Gram's method and Bismarck brown. Many of the yeast cells are decolorized and stained with Bismarck brown. x50.

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[19] Report of Working Party No. 1, etc., p. 29.

[20] Accompanying the residual food supply, as indicated by Nuttall and Shipley, "Studies in Malaria," Jour. of Hygiene, vol. iii, No. 2, April, 1903.

with smears from a culture of a white yeast kept in the laboratory. A week or so later I showed these slides to Drs. Parker and Rosenau of the Marine-Hospital Service and expressed the opinion that the fusiform bodies were yeast cells.

In the endeavor to account for the presence of these bodies in our non-infected mosquitoes and their absence from those that had been infected, it was recalled that the insects had been reared and kept in different buildings and fed differently for the greater part of the time at least. The non-infected mosquitoes were supplied with banana; the infected insects referred to in this paper were at no time permitted to feed on fruit, but were given only sugar and water in separate vessels. Many of the infected insects were reared under these conditions. I can speak positively on this point, because from the establishment of Camp Lazear until its abandonment the infected mosquitoes kept there were under my exclusive charge and control.

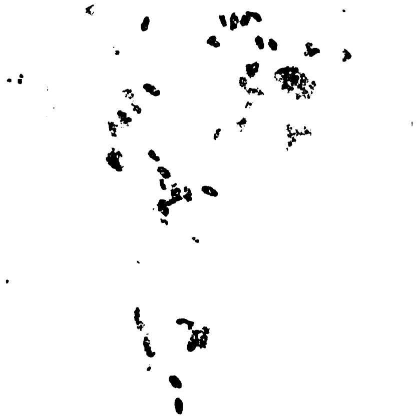

Fig. 6.—The same. A field from the diverticulum. x600.

To establish, if possible, the connection between overripe fruits and the presence of these bodies in mosquitoes, I obtained some *Stegomyiæ* through the courtesy of Dr. Edmond Souchon of New Orleans and Dr. L. O. Howard of the U. S. Department of Agriculture, the latter kindly supplying me with fifty living insects, a large number of eggs and some insects preserved in alcohol. Fusiform yeast cells were found in the diverticula of a very small proportion of those that were received in alcohol. An ellipsoid wild yeast (see Fig. 4) was then isolated from an old banana and insects were subsequently fed on the fermenting fruit, to which this yeast in pure culture had been added. Previous sterilization of the banana was not considered necessary. As a result, I have brought about the presence of bodies identical in appearance with the fusiform stage of the so-called *Myxococcidium stegomyiæ* in nearly fifty insects that have never been in contact with a case of yellow fever, and have demonstrated them in large numbers, in the diverticula of four *male* insects that have been hatched and reared in the laboratory of the surgeon-general's office in Washington. I have been able to demonstrate the passage of these organisms from the ventral diverticulum through the natural opening of communication to the esophagus and thence to the stomach, where they are found only in the upper part of the organ, and are absent from the blood mass below, which is of large size and recently ingested. I have obtained in one instance as the result of feeding a male insect on wild yeast and banana, sections which show these organisms among the muscular tissues of the thorax, many of them lying in close proximity to the gland. Several insects similarly treated show the same

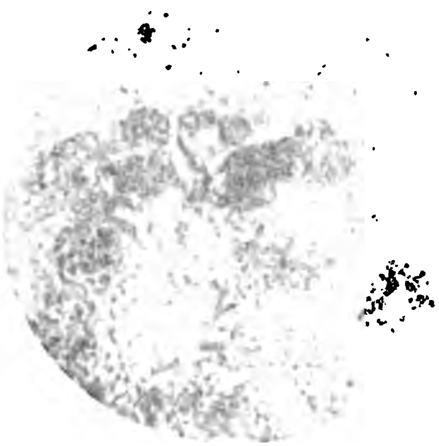

Fig. 7.—Colonies in the albuminoid mass in the diverticulum of a yeast-fed insect. Bismarck brown. x600.

bodies in the stomach and diverticula. (see Figs. 5, 6 and 7). They have not been found in fifty-two *Stegomyiæ* fed on yellow fever patients and kept under my immediate charge, a number of them having been used in reproducing the disease. Many of these mosquitoes that were kept for inoculation purposes died in the jars and are poorly preserved, but a number were killed and preserved immediately. The state of preservation of the insect, however, does not materially affect the staining properties of the blastomycetes, for in non-infected insects that have died in the jars and are crowded with bacteria, they color brilliantly and sharply. This would seem to indicate that they still retain their vitality after the death of the mosquito containing them.

Dr. L. O. Howard informs me that Dr. J. W. Dupree of Baton Rouge, La., writes him that he also finds the "*Myxococcidium stegomyiæ*" in non-infected mosquitoes at Baton Rouge.

Indirect confirmation of my results is seen in the report cited,[21] where it is stated that the parasite was not found in *Stegomyiæ* that had been applied to a malignant case of malarial fever and were then fed on blood, sugar and water. If a sufficient number of insects had been used and they had been fed on overripe banana or other fruit, as the working party recommends for yellow fever mosquitoes,[22] the supposed animal parasites would in all probability have been demonstrated.

On close examination of the liberated sporoblasts (?) shown in Figures 25 and 26 of the report referred to, it will be seen that these bodies show the variations in size and form and the typical budding process of yeast cells. This is particularly well marked in the left central and lower portions of Figure 26.

That ellipsoid wild yeasts may be pathogenic for the lower animals is shown by the researches of Rabinowitch,[23] who states also that these cells are stained by all the usual anilin colors. In the course of my own observations it has been found that typical ellipsoid forms in one of the mosquitoes were stained equally well with eosin, Bismarck brown, hematoxylin and by Gram's method.

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[21] Report of Working Party No. 1, etc., p. 30.
[22] Report of Working Party No. 1, etc., p. 28.
[23] Zeitschrift f. Hyg., etc., Leipzig, vol. xxi, 1896, p. 11.

Similar forms of blastomycetes have already been isolated from the broken-down nodules of spurious glanders, and the statement is made by Italian investigators that several parasites which are classified under the sarco- or micro-sporidia belong really to the blastomycetes.[24]

As the yeasts are universally disseminated in Nature (and there are many species of them, only a relatively small number being pathogenic), it is not surprising that experiments with a single species have failed to show positive evidences of invasion of the tissues of the insect. This was obtained by the working party of the Marine-Hospital Service in only a few instances, and is obviously of no importance from the point of view of the etiology of yellow fever. On the other hand, it is regarded as of the greatest importance that bodies identical with the fusiform stage of this supposed protozoön, the only form found to be constantly present. can be easily reproduced anywhere by feeding to mosquitoes a culture of an ellipsoid yeast.

The results of the observations here recorded are believed to justify the following conclusions:

1. The fusiform stage of the so-called *Myxococcidium stegomyiæ* of Parker, Beyer and Pothier (1903), is not connected in any way with the transmission of yellow fever.

2. This organism appears to be not a protozoan parasite, but a yeast fungus. In its fusiform stage, the only form in which it was constantly present, it shows the characteristic budding, staining affinities and vacuolation or spore formation of a blastomycete, and it is found with considerable regularity in both male and female mosquitoes that have purposely been fed on overripe banana to which a pure culture of a wild yeast had been added in the laboratory.

3. The organism has not hitherto been found in repeated examinations of mosquitoes of the genus *Stegomyia* that have bitten yellow fever patients in the early stages of the disease, when such insects had been fed only on blood, dry sugar and water. This statement applies also to mosquitoes that are known to have reproduced the disease in human beings.

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[24] "A New Pathogenic Species of Yeast," etc., Fermi and Aruch. Centralblatt für Bakt., etc., 1st Abt., vol. xvii, No. 17, May, 1895 p. 593.

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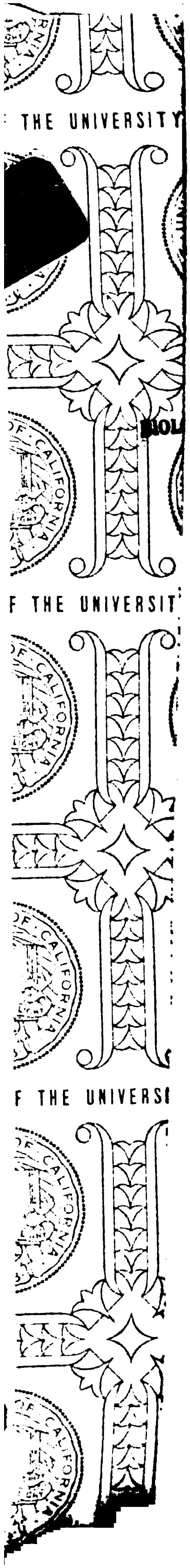
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rruccio, A.
ullettino della reale Accademia medica di Roma, - anno XII. -
Fascicolo 6°. - Giugno 1886.

LETTURE E COMUNICAZIONI

Prof. A. Carruccio. — **Sovra due casi d'inclusione di parassiti nematodi.**

I modi ed i mezzi d'introduzione dei parassiti nel corpo umano ed in altri organismi animali sono oltremodo diversi, e non di rado destano meraviglia nell'animo del medico o del naturalista che sappia e voglia studiarli.

Non meno maravigliose sono le fasi di sviluppo che essi percorrono, passando da una specie animale all' altra e le risorse con cui si mantengono o si adattano entro l'oste in cui riescirono a penetrare suo malgrado e le maniere diverse con cui poi si riproducono. Pel medico sono inoltre per lo più interessantissime le lesioni che sogliono accompagnare la presenza di larve o di forme adulte di parassiti ora entro un viscere, ora entro un altro, od anche sulla superficie della cute.

Dopo un lungo vagare per incerto cammino, oggi la Parassitologia, fondata su diligenti osservazioni ed esperienze sagacissime, trovasi in grado di rendere ragione di più e più fatti che dagli antichi scrittori giudicavansi inesplicabili, o se una spiegazione ne avevano tentata, questa so-venti oscurava vieppiù il modo di origine e di produzione di quei fatti.

I due casi, dei quali vi prego nell'odierna adunanza di prender conoscenza, sono appunto fra quelli che possono destare sorpresa, e sui quali la mente volentieri si ferma per intenderne il modo con cui poterono aver luogo.

Quando avevo la direzione dell'Istituto Zoologico della R. Università di Modena un distinto Medico dell'Ospedale Civile di Carpi mi scrisse annunciandomi di aver trovato un parassita entro un uovo di gallina; egual cosa mi fece, non ha guari, conoscere il chiarissimo collega ed amico professore Pietro Meloni, che insegna patologia generale nella R. Università di Cagliari. Il primo, ch' è il Dottor Grosoli così si esprimeva:

« Tengo nello spirito un corpo d'aspetto albuminoso, assottigliato alle estremità, lungo circa 15 centimetri, e della maggior larghezza d'un mezzo centimetro, ravvoltolato su se stesso, e con qualche striscia trasversale.

« L'ho guardato al microscopio, ma a parlarvi schietto, non ho capito nulla della natura di questo corpo; il quale ho trovato entro un uovo di gallina non covato. Giova sappiate che il tuorlo era sanissimo, e l'albume ridotto a poca quantità. Or bene, che ne dite? È l'albume rappreso che si presenta sotto quella figura, è un verme, o cosa è? A voi l'ardua sentenza... Se credete che meriti un esame diligente, ve lo posso inviare; se no, no (1). »

Avendo io pregato il Dott. Grosoli di mandarmi il corpo da lui annunciatomi, egli subito e gentilmente lo trasmise per mezzo dello studente del 6° anno della Facoltà Medica, sig. Benassi, insieme ad una lettera in cui mi dava la' seguente notizia:

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(1) Lettera del 6 novembre 1872.

« Debbo dirvi che una delle galline che hanno fatto le uova, fra cui trovai quello contenente il corpo che vi mando per istudio, è morta. Se ne fossi stato avvisato in tempo, l' avrei sezionata. » (1) L'egregio collega rispondeva con ciò a una mia domanda.

Il verme di cui è parola fa oggi parte della collezione elmintologica che fu da me istituita di pianta nel R. Museo Zoologico di Modena: questo verme era un *Heterakis inflexa.*

Alla sua volta il professor Meloni - Satta così di recente scrivevami: (2)

« In una sera del marzo 1885 la gentildonna Cagliaritana N. N, facevasi apprestare un uovo regalatole la mattina stessa da una gallina del suo pollaio. L'uovo però non era cotto: era appena portato al grado di trasudamento.

« Rotto il guscio, la signora se lo appressò alle labbra per sorbirlo. Ma, fu tutt'uno, impallidire e venirle la nausea ed il vomito... L'agitarsi convulso ed il saltellare agilissimo nell'uovo di un parassita, ne era stata la causa.

« Calmata la prima impressione di spavento, la signora e quei di casa si accertavano della presenza, nell'uovo, di un vermicciattolo lungo, lungo. Sorpresi del fatto singolare, unico e nuovo per loro, si ebbe la previdenza di custodirlo collo

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(1) Lettera del 9 novembre 1872.

(2) Lettera del 16 giugno 1886.

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stesso uovo, che la mattina susseguente veniv a me consegnato. Il vermicciattolo, quantunqu privo di movimenti, pareva vivesse ancora, e appariva fusiforme in mezzo alla vitellina Estra tolo dopo una mezz'ora, volli tener conto dell dimensioni: misurava 70 millimetri di lunghezz ed un millimetro di larghezza massima.

« Nulla di anormale trovai nel guscio, nè pe grossezza, nè per forma, nè per colore. Era u uovo normale in tutte le sue parti. L'albume e l vitellina normali anch'essi all'occhio nudo. Il lor esame microscopico avrebbe forse rilevato qualcosa d'importante, o quanto meno fatto pales se altro genere di parassita contenessero. Ma al momento, avendo fissata tutta la mia attenzione sul vermicciattolo, non pensai a siffatto esame.

« Non conosco altri fatti consimili, e non mi è stato possibile rinvenirne nelle opere all'uopo consultate. A me pare interessantissimo e degno di nota. Te lo mando per mezzo dell'onorevole deputato N. N. onde tu voglia dirmi se sia un vero ascaride; quale lo mostra agli occhi miei il microscopio. Tanto meglio per me se tu lo troverai meritevole di una comunicazione a codesta illustre Accademia. Il vermicciattolo io l'ho sempre conservato nella glicerina.

« Cercare il pelo nell'uovo è un antico e nuov adagio, che si ripete quando certuni amano so tilizzare o sofisticare su argomenti e questio piane e facili che non dovrebbero dar luogo a di scussioni o interpretazioni varie. Ma se non d vesi aver timore per la presenza di un pelo ne l'uovo, domando io se inquilini assai più moles e temibili, che osano sfruttare oltre l'allogg gratuito, anche i materiali nutritivi entro l'uovo, col passaggio del contenuto dell'uovo medesimo nel nostro organismo, possano essere per avventura di talun disturbo fisico, come di morale lo fu certamente nella egregia gentildonna di cui ti ho fatto menzione. »

Accogliendo il desiderio espresso dal chiaro collega ed amico prof. Meloni Satta, cioè di comunicare all'Accademia nostra il fatto da lui riferitomi, se stimavo che questo fosse meritevole di studio e di speciale comunicazione, darò oggi volentieri all'Accademia stessa la risposta. Ed in pari tempo quantunque con involontario ritardo, risponderò pure al bravo collega Carpigiano.

Premetto che anche nei Gallinacci molti sono i parassiti che possiamo trovare innicchiati in organi diversi, e taluno di essi riesce assai dannoso. Assai difficile però è il caso di trovare esseri animali o vegetali entro il contenuto dell'uovo delle Galline; ed anche leggendo opere speciali molte, difficilissimo riesce di trovar citati casi identici a quelli osservati dal Dott. Grosoli e dal professore Meloni Satta.

Dei parassiti vegetali rinvenuti dentro uovo di gallina ricorderò il fatto interessante narratoci dai prof. B. Grassi e Corrado Parona, osservato in Pavia nel gennaio 1878. Essi trovarono presso il tuorlo di uovo, che macroscopicamente e microscopicamente nulla presentava di anormale, un corpicciuolo aderente (ovicino fecondo) lungo 12 millimetri parzialmente involto da membranelle incomplete: anomalia molto rara è questa, nota col nome di *Ovum in ovo.* Senza ripetere tutti i particolari opportunamente esposti dagli autori, ricorderò come l' albume dell' ovicino apparisse composto di una quantità sterminata di *elementi morfologici*, che per tutti i loro caratteri fisici e chimici furono giudicati spore; le quali coltivate colla solita camera umida, in ambiente piuttosto caldo, dopo 10 giorni germinarono e si svilupparono moltissimi *Leptomitus* (1). Aggiungerò che già prima (1870) altri *Leptomitus* aveva trovati il Dottor Achille Fumagalli in un uovo di gallina (2).

Altri autori citano esempi di critogame trovate nell'interno delle uova. Rammenterò, fra le altre, le seguenti specie:

Sporothricum albuminis Burdach
» *brunneum* Schenk
Dactylium ovogenum Rayer
Hœtophora... spec. Hoffmann
Mucedinee... Panceri

Giova sapere come i predetti colleghi, i professori Parona e Grassi, abbiano – studiando questo argomento - sostenuto l'ipotesi della *Plasmogonia*, e per spiegare la presenza del gran numero di spore da loro trovate, così invero hanno scritto, cioè che « la composizione chimica dell' albume dell'ovicino era capace di dar luogo alla formazione di spore. »

E basti questa brevissima notizia intorno a corpi di natura vegetale rinvenuti entro uova di gallina, e de' quali non è mio intendimento, nè sarebbe il luogo, di qui occuparmi. Permettetemi invece altre brevi notizie intorno a diversi altri strani rinvenimenti di corpi, pure inclusi in ova di gallina.

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(1) Rendiconti del Reale Istituto Lombardo: Volume XI, pag. 237-245, 1878, Milano.

(2) *Sopra un microfito trovato in un uovo integro di gallina.* Rend. del R. Istit. Lomb. - S II. Vol. III. 1870, pag. 196

I professori Grassi e Parona giustamente osservano come debbasi confessare che questi casi sono piutosto rari. Davaine (1) cita la presenza di uno spillo; De Murs (2) ricorda uova d'uccelli nello spessore del cui guscio si trovarono frammenti d'insetti (un caso di Rogier ed uno di Moquin-Tandon); ma piuttosto che supporli passati dalla cloaca nell'ovidotto, senza « che quelli insetti si trovassero al luogo dove durante l'ovificazione posava l'ano dell'uccello e, sorpresi nella materia calcarea ancora molle, dibattendosi vi saranno rimasti incrostati in parte od in totalità; ed il raffreddamento quasi istantaneo di quella materia ve li avrà quasi inchiodati dentro. »

« Secondo Pouchet (citato da Panceri) zampe d'insetti ascesero dalla cloaca nell'ovidotto per mezzo delle loro spine, rivolte tutte per un senso. »

Il *Distoma ovatum*, che abita la borsa di Fabricio, nella gallina, può passare nell'ovidotto (e vi fu riscontrato da Otto, e può trovarsi nell'uovo (Hanon, Purkinje, Eschholz, Schilling, Gurlt).

L'ipotesi della penetrazione de' corpi stranieri attraverso la normale comunicazione della cloaca coll'ovidutto fu sostenuta dal professore Panceri con valide ragioni (3), ed è pur ammesso che l'ovidutto compie movimenti antiperistaltici, attissimi, perchè dal basso in alto, ad operare una specie di assorbimento, o succhiamento come meglio potrebbe chiamarsi, non solo dello sperma all'atto del coito, ma di corpi stranieri che vengano ad eccitare lo sfintere della cloaca.

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(1) Mem. sur les anomalies de l'œuf. Compt. Rend. de la Societé de Biologie. Ser. 3, T. 2, 1860. Vedasi inoltre le memorie dei professori Grassi e Parona, con queste citazioni, nei predetti Rend. dell'Ist. Lomb.

(2) Traité d'ovologie. Paris, 1861.

(3) PANCERI. — *Intorno ad alcune crittogame osservate nell'ovo di struzzo.* Vol. VI, degli atti della R. Accad. delle Sc. fis. e nat. di Napoli. 1873.

E posto anche che il muco, o l'epitelio vibratile per la direzione con cui è disposto sulla mucosa dell'ovidutto si oppongano all'ascesa del parasita, questo coi suoi movimenti vivacissimi, colle sue contrazioni ripetute, vincer può assai bene l'ostacolo che gli si oppone, e raggiungere la parte dell'ovidutto dove per la formazione del guscio calcareo rimane, volere o non volere, imprigionato.

Intanto, per meglio renderci conto di questi fatti conviene che diamo in forma riassuntiva uno sguardo alla struttura, alla situazione ed alla fisiologia dell'apparato ovarico nella gallina. A tal uopo, oltre questa eccellente preparazione che vi presento (1) vi prego di esaminare la diligentissima tavola murale che ho fatto disegnare e colorire allo studente signor Giulio Alessandrini, la cui abilità artistica è ormai ben nota.

Il riassunto che fa al caso nostro ce lo apprestano i precitati Parona e Grassi, e di esso possiamo valerci tanto è l'analogia dei fatti.

In vero, è noto che quando il vitello è maturo, la sua capsula viene abbracciata dall' estremità dilatata dell'ovidotto; si rompe e l'ovo sfugge nell'ovidotto col grand'asse diretto parallelamente all'asse del condotto e il disco germinativo per conseguenza trovasi respinto sul fianco. Nella discesa, attraverso l' ovidotto, si formano le parti accessorie dell'uovo.

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(1) L'apparato ovarico, nel suo pieno sviluppo lo si preparò in una gallina di mole straordinaria, donata testè al Museo dal Sig. Filippo Traverso di Roma.

Del pari è noto che l' ovidotto viene distinto dagli anatomici in 4 parti: la prima è imbutiforme; La seconda lunga, tubulare, s' apre per un condotto stretto, od istmo; nella terza parte, ch' è ampia; La quarta angusta, conduce nella cloaca.

Gli involucri del tuorlo si formano esclusivamente nel 2° e 3° tratto: nel secondo si forma l'albume. Tra il 2° od istmo ed il 3° tratto si forma la testacea. Nel 3° infine si forma il guscio. È finalmente noto che l'uovo arriva nel 3° tratto in meno di 4 ore, e che qui si ferma da 12 a 14 ore.

Ciò premesso, osservano i professori Parona e Grassi si può ragionevolmente supporre che il tuorlo dell'ovicino, prima che si distaccasse dall'ovario l'uovo contenente il corpo estraneo, sia entrato nelovidotto e sia disceso nell' istmo; lungo questa strada man mano sarebbe stato ravvolto dall' albume; nell'istmo avrebbe ricevuto una testacea. Ed a me pare che tale supposizione dei sullodati colleghi sia perfettamente ammissibile.

Intorno all'accennato differenziamento, che corrisponde alla diversità di funzione delle singole porzioni dell'ovidutto, mi piace aggiungere qualche maggiore particolarità, a complemento di quanto scrissero i precitati colleghi. La porzione anteriore più lunga e larga, cioè con ampia apertura imbutiforme, è quella in cui si secerne l'albume, il quale avvolge il vitello o tuorlo, ed a questa porzione, fornita di ghiandole tubolari, può darsi il nome di *tratto albuminogeno.*

La porzione media (che Gegenbaur chiama meno esattamente posteriore) più larga ancora della precedente, ma più breve, ovoide, disposta quasi a serbatoio, a superficie interna villosa, è quella dove si produce il guscio, che dapprima ha apparenza di fluido bianco, e poi gradatamente ma presto si solidifica: a tale porzione può serbarsi il nome di *tratto conchillifero.*

Finalmente la porzione ultima e posteriore dell'ovidotto sinistro, corta, stretta, è quella che direttamente sbocca nella cloaca accanto all' uretere sinistro.

L'ovidotto sinistro non solo è ben sviluppato- perocchè l' altro corre la sorte dell' ovaio destro che diventa atrofico, - ma negli uccelli è anche complicato: infatti ha ghiandole speciali situate in diversi punti della sua parete interna, e destinate a secernere o l'albume, come si disse, o le membrane, o il guscio dell'ovo; guscio che offre diversità moltissime sia pel volume, sia per lo spessore delle pareti, sia per la colorazione generale o predominante, o anche per le macchie variabilissime.

Le fibre muscolari dei diversi tratti sono disposte tanto nel senso longitudinale, quanto nel trasversale: e tale disposizione giova evidentemente alle funzioni cui essi tratti sono destinati.

Tutto ciò io doveva premettere per poter più agevolmente rispondere al seguente quesito:

Tanto l'uno, quanto l'altro parassita che mi furono comunicati, ebbero a svilupparsi nell'ovidotto, oppure nell'intestino?

Io penso che il parassita si è sviluppato nell'intestino, e poscia passò nell'ovidotto, dove potè ingrandirsi perchè alimentato dall'albume, rimanendovi incluso.

Tutto porta a credere che esso parassita provenisse dall'intestino, e che disceso fino all'ultima porzione rettale, e precisamente alla cloaca, penetrasse nell'orificio - che poteva essere dilatato per l'espulsione di altro ovo di recente avvenuta - penetrasse, dicevo, nell'orificio dell'ovidotto.

Ma ecco che può farmisi quest'altra domanda:

Il parassita è penetrato pel cavo peritoneale?

A siffatta domanda, che pur mi son rivolta io stesso, ma alla quale altri potrebbe rispondere con maggior convincimento di quello che sia in me, debbo ad ogni modo una risposta.

Ammesso per un istante che l'ascaride fosse penetrato nella sua parte più larga ed imbutiforme, sarebbe dovuto passare attraverso un'apertura intestinale. È però ammissibile, od almeno è facilmente sostenibile la penetrazione di un nematode non piccolissimo nel cavo peritoneale?

Non nego la possibilità del fatto, perocchè conosco anch'io casi bene accertati di ascaridi trovati nel detto cavo peritoneale; ma in questi casi sempre trovossi infiammato l'intestino, e all'infiammazione avendo tenuto dietro soluzione di continuità, cioè fistola cancrenosa, per questa poterono passare dall'intestino nell'ovidotto uno o più parassiti animali o vegetali, od altre sostanze, come ci viene dichiarato da Larcher e da altri autorevoli scrittori. (1) Il Larcher scrive: « Talora anche la parete intestinale viene aggredita da alcuni entozoari, i quali giunti a perforarla ed a collocarsi al di sotto della sua tonaca esterna, possono attraversare, a sua volta, eziandio quest'ultima e continuare la loro migrazione fino nel cavo peritoneale. »

Dei due fatti a me riferiti dal dott. Grosoli e dal prof. Meloni Satta, può credersi che nel primo la gallina abbia soccombuto alle conseguenze dell'elmintiasi; infatti il dott. Grosoli accenna nella seconda lettera scrittami alla morte di una delle galline, che avevano deposto uova, e fra queste trovossi quello contenente l'ascaride inviatomi al Museo di Modena. Il prof. Meloni Satta, nulla avendomi scritto intorno a fenomeni morbosi presentati dalla gallina, che depose l'uovo racchiudente il parassita, puossi credere che essa gallina fosse e sia rimasta sana.

I caratteri del parassita trasmessomi dal professore Meloni-Satta, e che vi presento, lo fanno annoverare nel genere *Heterakis* Dujardin, che distinguesi dai veri Ascaridi (Gen. *Ascaris* L.) per la presenza negl'individui maschi d'una ventosa preanale e di due spiculi per lo più ineguali; inoltre trovansi almeno tre papille preanali da ciaschedun lato.

I caratteri poi specifici, dopo fatta una conveniente diagnosi differenziale, fanno riconoscere l'individuo trasmessomi come appartenente alla specie chiamata già dal Bloch *Heterakis papillosa* (1).

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(1) Dott. O. Larcher: *Memoria sulle affezioni delle parti genitali negli uccelli.* Vedi Gazzetta Medica Veterinaria. Milano, 1874. Anno IV, pag. 240-243. - Vedi pure altra memoria del dott. Larcher: *Sui corpi estranei delle vie digestive degli uccelli.* Gazzetta predetta, 1872, pag. 596-601.

Non mi è stato facile, come dissi in sul principio, di trovare altri casi narrati in opere italiane che s'identifichino con quelli del Grosoli e del Meloni Satta. Forse, dedicandovi maggior tempo di quello che davvero non mi fu concesso in questi mesi di molte occupazioni, sarà dato a me stesso, o ad altri più fortunati, di riunirne parecchi. Vi dirò solo che nel *Giornale di Medicina veterinaria pratica*, dell'Accademia Reale Veterinaria Italiana in Torino, del 1880 (fascicolo 5, anno XXIX, pag. 318) trovai scritto quanto segue: « . . . . . Il Segretario generale presenta (seduta delli 31 marzo 1880) un verme stato inviato dal socio Zambelli, il quale viene riconosciuto per un ascaride, e che il detto Zambelli diceva, in seguito all'asserzione della sua domestica, essere stato rinvenuto in un uovo di gallina. »

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(1) Il Rudolphi, per limitarci ad un solo de' più competenti scrittori, dà la seguente diagnosi dell'*Ascaris vesicularis* Froel. (Sinonimo di *H. papillosa* Bloch): « *linea corporis laterali tenuissima: cauda utriusque sexus reflexa, in maribus utrinque basi connivente alata.* « Ved. *Entozoorum sive Vermium intestinalium Historia Naturalis.* Vol. II, P. I. Pariis ec. 1810, pag. 129. Ed a pagine 130-132 dell'istessa opera il Rudolphi aggiunge una minuta descrizioné delle varie parti del corpo del ⸸ e della † di questo nematode.

Importanti sono gli studi del compianto prof. Ercolani intorno al modo di sviluppo e di riproduzione dell'Ascaride vescicolare, oltre altre specie che pure ha studiato ed indica. Ved. sulla dimorfobiosi o diverso modo di vivere e riprodursi sotto duplice forma di una stessa specie di animali. - Vedansi pure le osservazioni fatte sopra alcuni Nematelminti - Bologna, 1873-1875. V. inoltre PERRONCITO: I Parassiti dell'uomo e degli animali utili - Vallardi. Milano1882 pag. 305-307

« Il presidente (prof. cav. R. Bassi) crede alla possibilità della presenza di tal verme nell'uovo, perchè rimontato dalla cloaca prima della calcificazione del guscio; e dice riservarsi di vedere se sianvi fatti identici anteriori. »

Null'altro più trovo detto intorno a questo fatto. Ma nel fascicolo di giugno 1880 dell'istesso giornale, a pag. 416-417, fu pubblicata una lettera del dott. Odoardo Benci di Calci, nella quale, a proposito del fatto del dott. Zambelli, così scrive: « . . . A me pure accadde di vedere il 2 maggio ultimo scorso un simile fatto di un ascaride, cioè, che rinvenni in un uovo di gallina, di razza nostrale, nuotante nell'albume e della grandezza naturale...

« A me sembra adunque, continua il dott. Benci, che questi due fatti, sebbene apparentemente isolati, si possano verificare con una certa maggiore frequenza. Solo che, invece di accadere sott'occhio a persone della scienza che vogliono darsi cura di renderli noti, è possibile il più spesso accadano sotto l'osservazione di persone volgari, non curantesi della cosa, ecc. ».

E qui potrebbe anche aver ragione il dott. Benci; ma nè questi, nè il Zambelli punto pensarono a spiegarsi il fatto da essi comunicato alla R. Accademia Medica Veterinaria; nè in questa mi risulta che, secondo la promessa del chiarissimo prof. Bassi, siano stati riferiti casi anteriori e consimili a quelli del Zambelli e Benci.

Permettetemi, prima che dia fine alla mia breve comunicazione, che vi presenti un elenco de' principali parassiti trovati nelle galline.

CLASS. **Nemathelminta.** - Ord. *Nematodi.*

I. - Fam. *Ascaridae.*

Gen. *Heterakis* Duj.

1. *H. papillosa* Bloch (nel cieco) *Ascaris vescicularis* Froelich.
2. *H. inflexa* Zed. (intestino).
3. *H. compressa* Schn. (idem).

II. Fam. *Strongylidae.*

Gen. *Syngamus* Sieb.

4. *S. trachealis* Sieb. (nella trachea). (1)

III. Fam. *Trichocephalidae.*

Gen. *Trichosoma* Rud.

5. *T. longicolle* Rud. (Intest. crasso e specialmente nel cieco).
6. *T. annulatum* Mol. (intest.).
7. *T. collare* V. Linstov. (idem).

IV. Fam. *Filaridae.*

Gen. *Dispharagus Duj* et Mol.

8. *D. nasutus Rud.* (gozzo).
9. *D. spiralis Molin.* (esofago).

CLASS. **Plathelmintha.** - Ord. *Trematodi.*

Gen. *Distoma.*

10. *D. ovatum* Rud. (crasso-retto).
11. *D. lineare* Zeder (idem).
12. *D. dilatatum* Miram (idem).
13. *D. armatum* Molin (idem) ed altra specie.
14. *D. commutatum* Dies. (idem).

Dell'ordine *Cestodi* si conoscono diverse specie di Tenie e taluna di Botricefalo. (Ved per il ri·

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(1) Trovandomi nel passato luglio 1886 in Torino ho potuto osservare, mercè la gentilezza del Dott. A Carità, Assistente del Chiar. prof. Perroncito, le fasi di sviluppo che egli riusci ad ottenere del *Syngamus trachealis;* e di questo parassita - che da un solo cattivo esemplare era rappresentato nella nuova collezione che vo formando nel Museo di Roma - m'ha favorito l'istesso Dott. Carità più individui †, e ⸸. Di tutto gli rendo ora nuove grazie.

portato elenco l'opera dei professori Rivolta e Del Prato – Ornitojatria – Pisa, 1879).

Il prof. Landois di Münster calcola che nell'interno delle uova di gallina possano trovarsi molte specie di vermi (26), de' quali 13 appartenenti ai nematodi, viventi ora nell'intestino dell'istessa gallina, ora nello stomaco, ora nella trachea, ecc., 8 ai trematodi, e 5 ai plattielminti (Tenie e Botriocefali).

Dal Landois, come pure da tutti gli scrittori competenti, affermasi che se taluni di questi parassiti penetrano nell'uovo di una gallina, questo uovo non può riuscir nocivo a chi lo mangia se prima fu sottoposto alla cottura. E bene s'intende tale innocuità delle uova, che tanto spesso servono quale ottimo alimento, quando si pensi che per l'azione del calore il parassita rimane od estinto affatto, o talmente alterato da finir di soccombere nel tubo digerente di chi lo ha deglutito col contenuto dell'uovo. Adunque anche se maturi, i parassiti inclusi entro uovo di gallina o d'altri uccelli ecc. - se questo uovo fu sottoposto a un grado di temperatura conveniente - non potranno riprodursi nel l'intestino umano. E ciò dico in risposta a una cortese domanda che mi fu fatta.

DISCUSSIONE.

Il Presidente dice che delle due ipotesi enunciate dal disserente, la prima è molto plausibile, mentre non sa se tutti gli Accademici potrebbero ammettere l'altra dal punto di vista anatomo-patologico.

Il Socio **Carruccio** ringrazia il Presidente delle considerazioni fatte, ma osserva che egli ha già ammesso la seconda ipotesi, come la meno probabile, mentre non può venire assolutamente esclusa, inquantochè per molti parassiti è ammessa la possibilità del passaggio attraverso le tonache intestinali.

Il Socio **Durante** concorda col presidente nell'escludere il passaggio diretto dei parassiti dall'intestino all'ovaia, inquantochè l'animale morirebbe di peritomite settica. Pensa piuttosto che per un lento processo flogistico, un'ansa intestinale possa saldarsi al tubo ovarico ed attraverso questa saldatura, in seguito a processo ulcerativo, i parassiti possano giungere nell'ovaia.

Carruccio replica, esponendo le ragioni per le quali non crede ciò possibile, pur avendo egli stesso discusso se il fatto esposto dal collega Durante possa accadere nell'organismo dei gallinacei. A tal'uopo ricorda la conformazione e la situazione del sistema riproduttore, e particolarmente dell'ovaio destro ricoperto nelle galline dal polmone, ed aggiunge che da una seconda lettera del Dott. Giustiniani Grosoli, apprende che la gallina osservata, era morta al secondo giorno dalla fetazione dell'uovo in discorso, ma non fu praticata l'autopsia: l'altra gallina era sana.

Osserva inoltre come in molte opere e giornali si leggano casi di enterite ulcerativa con accumulo di parassiti e loro passaggio attraverso le pareti intestinali.

Dott. E. Tassi. — **Estirpazione d'un fibromioma sessile delle pareti vescicali seguita da guarigione.**

Voi, o signori, sapete che la storia dell'arte chirurgica ha registrato pochissimi e rari casi di estirpazione metodica dei tumori delle pareti vescicali e di quelli esistenti nell'interna sua cavità di qualunque natura istologica siano formati. Nè potete disconoscere le difficoltà che essi offrono alla mano chirurgica più ardita e gli esiti sconfortanti che il più sovente se ne traggono. Di guisa che i più abili chirurgi fino a pochi anni or sono si rifiutarono quasi sempre di aprire la cisti urinaria per cosifatta estirpazione, anzi giudicarono codesto atto operativo proposto dal Platner e dal Warren come ineseguibile. Oggi tuttavia, in virtù dei mezzi antisettici la chirurgia volle trionfare eziandio in questa ardita operazione; e noi in Italia già possiamo contare qualche fatto che onora l'arte nostra. E adesso permettete a me, che come contributo a codesta splendida ed ardua operazione io ve ne presenti un caso perfettamente guarito: che forma il soggetto della storia seguente appositamente redatta da uno dei miei assistenti, il signor Dottor Cimbali.

Storia clinica. — La donna è nativa d'Anguillara, ha 25 anni, il padre vivente e la madre morta di malattia di cuore, inoltre ha un fratello e due sorelle che godono perfetta salute. Anche essa è stata sempre bene, menstruata a 14 anni ha seguitato ad avere la menstruazione tutti i mesi. È maritata da cinque anni ed ha due figli viventi e sani. Racconta che quattro anni e nove mesi fa, tre mesi prima di prender marito, in seguito a lavori faticosi di

F. H. J.
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Carter, H. J.
1878

Parasites on the Spongida.

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"RESPECTIVELY."

Pe [illegible]

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Dr. Wallich ·
with Mr Kind regards.

H. J. Carter,
The Cottage,
Budleigh Salterton,
DEVON.

STORY *for*

In 1871 ('Annals,' vol. viii. p. 330) I stated that I hoped soon to communicate an "illustrated paper on the parasites of sponges;" and now, after having examined all the specimens of the latter in the collections of the British Museum together with those belonging to the late Dr. Bowerbank, and with my own experience of the living sponges here (Budleigh-Salterton), I propose to notice those parasites which have come under my observation and of which I possess specimens, being well aware that there must be many more which have not been discovered, or, if discovered, have not been made public.

For illustrations I prefer figures which combine that of the sponge with that of the parasite; and therefore reference will be made to these whenever possible, while the rest hardly require any; so that the only illustration that I shall insert will be one of *Spongiophaga communis*, which will be given in a woodcut opposite the description.

Crustaceans.

It seems not uncommon for small Amphipod Crustaceans about 1-12th inch long to nestle in the surface of some sponges, where they make little oval depressions to lie in, more or less bent upon themselves, which depressions, in the absence of the crustaceans, may sometimes be taken for vents. This was first noticed in *Suberites antarcticus*, MS. (a branched Suberite of a grey colour, with large and almost spherical head to its pin-like spicules, dredged up by Sir J. Ross in 300 fathoms in 77½° south latitude), and the crustacean kindly described and illustrated by the Rev. R. R. Stebbing, M.A., under the provisional name of *Dexamine antarctica* ('Annals,' 1875, vol. xv. p. 184, pl. xv. fig. 1, &c.). Similar depressions with a smaller crustacean of a like form were afterwards observed on the surface of a large mouse-coloured, areniferous, estuarian variety of *Suberites domuncula*, Nardo, = *Halichondria suberea*, Johnston, on a *Buccinum* containing a *Pagurus*, probably from the Firth of Forth, Scotland, and, lastly, though of larger size, on a living specimen of *Halichondria incrustans* from this place (Budleigh-Salterton).

Crustaceans are commonly found in the cloaca and halfway through its aperture in *Grantia ciliata* and *G. compressa*, especially towards the maturity of the *gastrula*, which, being free from spicules and rich in nutriment, they devour greedily, not refusing portions of the sponge itself; so that, in gathering pieces of the seaweed on which *G. compressa* chiefly grows here, it is desirable to free the specimens as much as possible from the Alga, lest, under confinement, the crustaceans issue from their nests in the latter, where they dwell in great abundance, make an onslaught on the *Grantias*, and destroy the greater part of them.

CIRRIPEDES.

The Balanoid Cirripedes, whose embryos are so abundant that they almost cover every thing on the rocks here, together with the rocks themselves, could hardly be expected to refuse the surface of the Spongida; and hence, perhaps, they are the most common parasites of all; for, with the exception of the fleshy sponges (*Carnosa*) and the calcareous ones (*Calcarea*), they make use of every other kind of sponge, becoming, as they increase in size, overgrown by the sponge itself, whether the latter is kerataceous or vitreous, so as to form wart-like excrescences with a hole in the summit for the projection of the cirri. The species appear to vary in the same as well as in different localities; and the term "*Acasta*" has been applied to the whole group by Leach.

×

ACTINOZOA OR POLYPS.

In all parts of the world sponges are more or less infested by polyps, chiefly on the surface, which may be single, double, concatenated, or grouped, isolated or aggregated, sunk to the level of the surface of the sponge which they may infest without scleroderma, or with it *in* the scleroderma *on* the surface of the sponge, or pendent from the scleroderma; and all belong to the Zoanthidæ = Palythoa, Lamour., = Zoantha of De Blainville.

Of those on the sponges of the Antilles, Duchassaing de Fontbressin states:—

"Les Zoanthes, les Mamillifères et les genres voisins sont littoraux; cependant il y a des exceptions pour quelques-uns des ces êtres, comme le *Zoanthus parasiticus*, le *Gemmaria Swiftii* et les *Bergia*, qui toutes sont parasites des éponges, et que j'ai recueillies par une profondeur variant entre 2 et 8 mètres. Ces espèces ne se trouvent jamais que fixées sur les Spongiaires; elles ne se rencontrent sur aucune autre espèce de corps marins." ('Revue des Zoophytes et des Spongiaires des Antilles,' par M. P. Duchass. de Fontbressin, 1870, p. 22.)

Such are the words of this naturalist, who, with M. (afterwards le Chevalier) G. Michelotti, published copiously illustrated works on the corals and sponges respectively of

× Here add minute Ascidians on the surface of an Esperia from the N.W. Coast Spain.

the Antilles and the Caribbean Sea, gathered by themselves alive and dead in these localities before the year 1864 ("Spongiaires de la Mer Caraïbe, par P. Duchass. de Fontbressin et G. Michelotti," Natuurk. Holland. Maat. Wet. te Harlem, 1864, vol. xxi. 4to).

The character of these polyps is to have their sclerodermic parts more or less charged with foreign bodies, viz. grains of sand and sponge-spicules entire and fragmentary, derived from the sponges of the locality generally, but chiefly from the sponge on which they may be situated. I can, of course, state nothing of the softer parts in their original condition, as my descriptions are taken from dried specimens; hence *this* information must be sought from other sources.

1. *Polyps single or isolated, scattered over the surface more or less generally, sunk to the level of the sponge, but marginated; about* 1-16 *inch in diameter.*

Especially observed in the genus *Tuba*, Duchass. de F. et Mich. (*op. cit.*, e. g. *T. digitalis*, pl. viii. f. 2), Rhaphidonemata, fam. Cavochalinida, groups 6–8, Cart. ("Notes Introductory to the Study of the Spongida," 'Annals,' vol. xvi. p. 141) = *Siphonochalina*, Sdt.; also in *Reniera fibulata*, Sdt. (Holorhaphidota, group 5. Fibulifera, Cart. *op. cit.* p. 178), from the seas between the Americas; also in *Axinella polypoides*, Sdt., from the Adriatic sea (Schmidt, Spong. Adriat. Meeres, Taf. vi. f. 4).

2. *Polyps single or concatenated, scattered over the surface more or less generally, sunk to the level of the sponge, but marginated; about the same size as the foregoing.*

See especially the genus *Thalysias*, D. de F. et M. (*op. cit.*), Holorhaphidota, group 3. Thalyosa, Cart. (*l. c.*). For a good figure see *Isodictya mirabilis*, Bk. (Proc. Zool. Soc. Lond. 1873, pl. xxviii. figs. 1, 6, 8), = *Thalysias subtriangularis*, D. de F. et M., 1864 (*op. cit.* pl. xvii. fig. 1), from the seas between the Americas. The name used by Dr. Bowerbank must be suppressed, as it was given long after that of D. de F. et M.; and that of "inhalant pocilla" applied by him to the polyps is a mistake, carried on from his description and figure of 1864 (Mon. Brit. Spong. vol. i. p. 278, pl. xx. f. 308). The description, however, faithfully illustrated by his artist Lens Aldous, records all that is necessary respecting the dried form of the polyp.

Should instances of circumscribed *inhalant* caliciform or tentaculiform areæ in sponges be desired, they may be found in *Grayella cyathophora* and *Cliona corallinoides* respectively, as represented in the 'Annals' (the former in 1869, vol. iv. pl. vii., and the latter in 1871, vol. viii. pl. ii.).

3. *Polyps single, double, concatenated or irregularly grouped; sunk into a scleroderma upon, but not into, the surface of the sponge; circumference of the polyp defined but not marginated, about* 1-12*th inch in diameter.*

See especially *Echinonema typicum*, Cart. MS. (Echinonemata, fam. Ectyonida, group 1. Pluriformia, *op. cit.* p. 143, &c.). From Freemantle, S.W. Australia. *Very* common on the branched digitate form.

4. *Polyps single, double, or irregularly grouped, more or less pendent from their scleroderma, situated upon the surface of the sponge; sometimes* 1-4*th inch long.*

Ex. gr. *Axinella damicornis*, Sdt., and *A. verrucosa*, Sdt. (Spong. Adriat. Meeres, Taf. vi. figs. 2 and 3 respectively, 1862). *Palythoa axinellæ* is Schmidt's name for this polyp, which is more pendent but smaller in the head than the following species, viz. *Palythoa fatua*, M. Schultze ('Hyalonemen,' 1860, S. 27, *ff*), which grows over the upper part of the glass cord of both *Hyalonema Sieboldii*, Gray, from Japan, and *H. lusitanicum*, Boc., from the Atlantic, on the coast of Spain and the north of Scotland. See *H. mirabilis*, Gray (Proc. Zool. Soc. Lond. 1857, = *H. Sieboldii*, Gray, 1835, *ib.*), partly copied into Dr. Bowerbank's 'Mon. Brit. Spong.' vol. i. p. 287, pl. xxxv. f. 374, where the polyps are considered by Dr. Bowerbank to be the "oscula" and *not* the "inhalant areæ" of the sponge, as stated and delineated in fig. 308, *ib.*, before mentioned! This somewhat ficoid species occurs on the depressed and sessile forms of *Tethea muricata*, var., Bk., = *Normania crassa*, Bk. (Mon. Brit. Spong. vol. iii. 1870, pl. lxxxi. fig. 1), where there is a group of four figured *without indication*, on the right side of the median line close to the upper margin, which I recognize here, especially, because the same thing occurs on a similar specimen dredged up on board H.M.S. 'Porcupine' between the north of Scotland and the Färoe Islands.

With reference, however, to Duchassaing de Fontbressin's statement before quoted, viz. that the parasitic polyps of sponges to which he alludes do not occur on any other marine organisms, there is, in the British Museum, a flat, elliptical, sessile mass or colony of *Hydroid* podocorynid polyps about three inches long and one tenth of an inch thick, whose delicate, erect, colourless filaments in juxtaposition, like the hairs of a clothes-brush, rising from a tough matted mycelium, present an even surface of hydranths on the top sufficiently firm to support several patches of a parasitic polyp, to me identical with the *Palythoa fatua* of the glass cord in *Hyalonema Sieboldii* &c.

On the other hand they are present at such an early period in some sponges that at first it seems as if they were part of the sponge itself, or, at least, developed in combination with it. But when we reflect on the unerring certainty with which the pollen-grains of diœcious plants find their way to the stigma of the female flower *through the air*, and, indeed, the spermatozoa of the myriads of beings, both animal and vegetable, growing together on our shores, find their respective species amidst hosts of others on the same errand, *through the sea*, we cannot wonder that a similar instinct directs the parasitic Zoanthidæ in their embryonic state to find the objects on which they respectively prefer to dwell. At the same time, as these polyps are not seen on the sponge at a very early stage of development, nor are always present on the same species, it is evident that they are not a part of the sponge, nor developed *pari passu* with it; while it is equally evident that, in the *first* instance, they must have come from an unparasitic *Palythoa*, and therefore have obtained their specific differences subsequently, although, when once these have been obtained, they continue, from adaptation, to prefer their new habitat to that of the original stock. This, indeed, is the law of adaptation and inheritance.

Hydrozoa or Hydroid Polyps.

While in all cases of Actinozoic parasitism that have come to my notice in sponges the polyps have been *confined to the surface*, those of Hydrozoic parasitism have extended into *the deepest parts of the sponge*, and, in one instance, have been entirely confined to the interior.

Taking, first, those whose tubes opened on the surface—one was found by Dr. Allman in a "horny sponge on the southern shores of France" and called by its discoverer "*Stephanoscyphus mirabilis*" (Trans. Linn. Soc. 1875, ser. 2, vol. i. pt. i. tab. xiv.; and 'Nature,' 1874, July 30, p. 251); and the other in *Reniera fibulata*, Sdt., *Suberites flavus*, Liebkh., *Esperia Bauriana*, Sdt., and *Myxilla fascicularis*, Liebkh., respectively, by Prof. F. E. Schulze in the Adriatic Sea, who designated it *Spongicola fistularis* (Archiv f. mikroskop. Anat. 1877, Bd. xiii. p. 795, Taf. 45–47); while the instance in which the Hydrozoon was confined to the interior of the sponge occurred to myself, and was noticed in a specimen of *Reniera* (*R. polypifera*, Cart. MS.) from Bona

Bay on the north coast of Africa ('Annals,' 1872, vol. x. p. 50).

Of *Stephanoscyphus mirabilis* Dr. Allman states that it "may be found attached to stones in small patches of one of the horny sponges," of which the figure in the Trans. Linn. Soc. (*l. c.*), being of the "natural size," is about two inches in diameter and half an inch thick in the middle. This consisted of a "congeries of tubes which penetrate the sponge-tissue and open on its surface," being, with their contents, "united by a common tubular plexus towards the base of the sponge" ('Nature,' *l. c.*).

On the other hand, *Spongicola fistularis* is stated by Dr. Schulze to consist of a series of branched tubes (*l. c.* Taf. xlvii. fig. 8), opening on the surface of the sponge by one end (Taf. lxv. fig. 1), and closed or blind at the other. Hence *Stephanoscyphus mirabilis* not only differed from *Spongicola fistularis* in this way, but the former being in a "horny sponge" seems to intimate, although the kind is not mentioned, that it was in a totally different order from all those bearing the hydrozoon so elaborately described and beautifully illustrated by Dr. Schulze.

In my own case, where the polyps were situated in the interior of the sponge, I have nothing to add beyond what has already been stated, 'Annals,' *l. c.* (for the specimen was returned, with all the rest of the sponges dredged up on board H.M.S. 'Porcupine,' to Sir Wyville Thomson last year, 'Annals,' vol. xix. p. 432), viz. that "the minute delicate polyps were seated in dilated cavities, apparently of the excretory canals, the disk or head of each polyp averaging 1-100th inch in diameter, and supported on a short neck, which ended in a little saccular prolongation that was sunk into the parenchyma or sarcode of the sponge, and charged in its walls, as well as in its tentacles, with thread-cells, &c." But that my object then was chiefly to show that the thread-cells observed by Eimer in *Reniera fibulata* and *Desmacella vagabunda*, Sdt., probably did not belong to the sponge, as subsequently confirmed by Schulze's observations (*l. c.* p. 799), I should probably have paid more attention to the structure of the polyp itself, which, however, from its minuteness, position, and exserted tentacles, might be inferred to have been a Hydroid rather than an Actinozoid polyp like that of *Palythoa*.

ALGOID PARASITES.

Seaweeds.

It is not an uncommon occurrence in some parts of the world for a seaweed to become a pseudomorph of a sponge (to use a mineralogical term), in which the latter, like a "dissolving view," may be observed (through different specimens) to yield gradually to the former, so that, at last, the seaweed not only assumes the shape of the sponge generally, but that of the form and position of the vents and every other part of the sponge saving the spicules, or foreign bodies of a like nature, which thus are often the only remaining evidence of the *kind* of sponge that has thus been pseudomorphosed.

I noticed this first in specimens of *Reniera fibulata*, Sdt., from Hong Kong, in the British Museum, wherein parts of the sponge itself still remained to prove what has been just stated; and since then several specimens have been added from the late Dr. Bowerbank's collection, that were obtained from Freemantle on the south-west coast of Australia—which led me to seek for the seaweed in Harvey's 'Phycologia Australica,' where I found it figured under the name of "*Thamnoclonium flabelliforme*," also from Freemantle (vol. ii. pl. 13).

The fan-shaped pseudomorphs in the British Museum represent the figure, and bear remains of the spiculation of this form of *Echinonema typicum*, Cart. MS., which is very common at Freemantle; another, that of a Suberite with pin-like spicules only (that is, without any flesh-spicules); and a third bears on its surface portions of the reticulated incrustation of foreign bodies characterizing many of the Psammonematous sponges.

Frequently, as stated by Harvey, on the more prominent parts of this parasitic seaweed may be observed little pedicelled leaf-like expansions or young fronds, which, when softened by soaking in water, present "tetraspores lodged in discoid nemathecia, in their substance;" and thus far the reproductive elements of this Alga have been discovered.

Red Alga parasitic in Halichondria plumosa, *Johnston.*

There is an amorphous Alga (apparently undescribed) which infests some specimens of *Halichondria plumosa* on this coast, consisting of a pseudofrondaceous expansion of carmine-red cells, which, pursuing in its growth the main branches of the skeleton, from the base to their termination on the surface of the sponge, finally produce a dark-brown-coloured, equally amorphous, wart-like fructification.

Its cells are irregularly globular, of a beautiful carmine colour, and held together by a gelatinous membrane, which not only grows upwards round the axis of the branches mentioned, but extends outwards laterally for some distance over the echinating spicules of which they are respectively chiefly composed, finally ending on the surface in a clathrate structure, which throws out small, irregular, wart-like, botryoidal masses of a black-brown colour (in sizes below 1-36th inch in diameter). The latter are composed of a crust formed of radiating columns of brown cells in juxtaposition (each column consisting of a transparent theca enclosing about a dozen), containing, or accompanied by, or both, globular tufts of branched short filaments of *red* cells mixed with paraphyses, the filaments being clavate from the enlargement of the cells towards the free ends, thus becoming terminally (?) sporiferous, much like those of *Hypoglossum Woodwardii* figured by Payer (Botan. Cryptogamique, 1850, p. 47, fig. 209). No tetraspores could be recognized; but where the pseudofrondaceous layer had left the sponge and spread itself over surrounding *Balanus*-shells, it presented somewhat the appearance of *Hildenbrandtia sanguinea.*

The red cells of the thallus are about 1-4000th inch in diameter, and the brown cells of the columns about a third smaller, while the terminal cells of the branched filaments in the "tufts" are the largest of all. The brown warty fructification (? nemathecia) is surrounded by a transparent membranous envelope; but this, as well as all the other structures that I have mentioned, can only be seen under the microscope in a fresh state, or on soaking in water, after having become dry. The red colouring-matter of the cells is not affected by drying, nor is it much altered by the addition of liquor potassæ.

As this Alga appears to be unnamed and undescribed, this can be best done by those who have given their attention especially to the subject.

Oscillatoria.

There is a Suberite with pin-like spicule only (that is, without flesh-spicule), which occurs on the rocks here a little above low-water mark, in small thin patches about half an inch in diameter, of a beautiful cobalt-blue colour; and when examined with a microscope the blue colour is found to be owing to the presence of innumerable short separate filaments of an Oscillatorian alga, which, answering to the description of the genus *Hypheothrix*, Kg., but with blue granules, from which the cobalt-blue colour of the sponge is derived, might be called "*H. cærulea.*" The filaments vary in length under 1-1500th inch, with a diameter of 1-12000th inch; and the colour fades much on drying, but does not altogether disappear.

Scytonema.

A species of this Alga with its germinating gonidia still retaining their dark yellowish-green colour, is abundant in a specimen of *Spongia otahetica* in the British Museum, about which there are no remains of sarcode; so that it was probably after the death of the sponge that this Alga took up its abode there. It is therefore only mentioned here to show that, in describing the parasites of sponges, the circumstances under which they occur should not be forgotten; otherwise much more may be set down than really belongs to such parasitism.

There are also destructive organisms which not only attack the horny parts of the skeleton but the spicules themselves of a sponge after death, such as have been described and figured in the 'Annals' for 1873 (vol. xii. p. 457, pl. xvi. figs. 8, 9).

Palmella spongiarum, Cart.

In two instances I have found at this place sponges which have been rendered pink by the presence of a little spherical cell in great abundance, about the size of the human blood-globule,—viz. one in a specimen of *Halichondria panicea*, and the other in a specimen of *Cliona celata*. And on examining it with a microscope, I find that one mode of reproduction is by duplicate division, and that it is enveloped in a mucilage, which, as the *Palmella* grows and the cells become multiplied, thus extends itself throughout the sponge, and, by the immense number of its cells, produces the pink colour. The colour fades to a certain extent, but not altogether, on drying, and is changed to green on the addition of liquor potassæ, when it becomes very like a green *Protococcus*. While retaining the pink colour, it has very much the appearance, under the microscope, of *P. nivalis*, but is much smaller. It averages 1-2400th inch in diameter; and, not being polymorphic (that is, not being able to change its spherical form), it cannot be confounded with the ovules of the sponge, especially when of this size.

? SAPROLEGNIEÆ.

Spongiophaga communis, Cart. 1871.

This is a minute, short, nematoid filament, with a bulb at each end, which, multiplying to an enormous extent especially in the *Hirciniæ* (Hirciniosa, 3rd Group, 'Annals,' *l. c.* p. 136), may, like the seaweed *Thamnoclonium flabelliforme*, become a pseudomorph of the sponge it attacks, so as to be mistaken for the sponge itself, as will presently appear.

In 1859 (Archiv f. Anat. u. Phys. Heft iii. p. 369, pl. x. fig. 2), Lieberkühn considered this filament to be a character of certain *Hirciniæ*, which he called "Filifera;" and in 1862, Schmidt (Spong. Adriat. Meeres, p. 30) accepted the character and proposed for the genus the following diagnosis:—

> "Ceraospongiæ duplici fibrarum genere præditæ, uno crassiorum, quæ inter se cohærentes sceletum proprie formant, altero subtilissimarum, quæ ex illis provenientes minutissimis capitulis terminantur et inter se non implicantur."

According to Schmidt (*op. et loc. cit.*), it was observed by Esper, who likened it to "wool;" but neither Esper nor Nardo made it a "character" of *Hircinia*.

In 1845 ('Annals,' vol. xvi. p. 407, pl. xiv. figs. 1–5) Dr. Bowerbank represented it as a "most remarkable character" in his genus *Stemmatumenia*; and in 1864, Duchassaing de Fontbressin and G. Michelotti partly founded their genus "*Polytherses*" upon this parasite, which they describe as "moniliforme," and figure with transverse *septa*, like the filament of an *Oscillatorium* ("Spongiaires de la Mer Caraïbe," *l. c.* pl. i. F, and pl. xii. fig. 5, &c. species).

In 1871 ('Annals,' vol. viii. p. 330) I stated that this filament was an Alga, and probably an *Oscillatorium*, which, from its frequently infesting sponges of different kinds in all quarters of the globe, I proposed to name "*Spongiophaga communis*;" further, it was then stated that "Schmidt (1862, Spong. Adriat. Meeres, and especially with figures in 1864, 1st supplement), after having given a great deal of attention to these filaments, which have a cell at one end and a spiral twist throughout, admits that they are different from the sponge-cell *par excellence* (*i. e.* the sponge-animal), and, after alluding to Kölliker's doubt in 1866, viz. whether it be a part of the sponge or a parasite, agrees in 1870 (Atlantisch. Spongienf.) with Kölliker, that the two structures, viz. the sponge-fibre and the fibrillæ, are different, finally ending with the expression that, after much trouble, he can state nothing further respecting the nature of the latter."

To this may be added Schmidt's opinion in 1878, at least, in a paper entitled "Die Fibrillen der Spongiengattung, Filifera, Lkhn.," of which he kindly sent me a copy in May last, viz. "Meine Angabe, dass die Fibrillen von Hornfasern entspringen, beruhte auf Täuschung" (pp. 661–2), and, further on, that all attempts to get out an "entire" fibril fail. But it will presently be seen, in the special description of *Spongiophaga communis* which I am about to give, that this has been accomplished, although probably owing to the specimen being more favourable for the purpose than any possessed by Dr. Schmidt.

Figures of the filament, so far as it was known, have been given respectively by Lieberkühn, Bowerbank, Schmidt, and, lastly, by Duchassaing de Fontbressin and Michelotti, whose "moniliform" or septate character, before noticed, partly led me to the idea that it was a species of *Oscillatorium*, which further investigation has not confirmed.

As before stated, this parasite chiefly, but not exclusively, attacks the *Hirciniæ* in all quarters of the globe, but becomes most remarkable when it has *entirely* replaced the sarcode in those great bowl-shaped specimens that come from the seas between the two Americas and from the southern coast of Australia respectively. The specimen represented by Duchassaing and Michelot (*l. c.*), viz. *Polytherses campana*, is not an uncommon form, wherein the "bowl" has not been completed; while there are large massive forms also of this species of *Hircinia*, and some from the neighbourhood of Cuba, which present *no* filament; but, lest it should be fancied that these might have belonged to a different species and *therefore* not to possess the filament, it might be stated that in the British Museum there are some "bowl-shaped" ones from Australia which present nothing but the original sarcode, and others nothing but the filament respectively covering their skeletons, which thus, in each instance, retain the "bowl-shaped" form of the original sponge.

Besides this, it is abundant in a specimen of *Axinella faveolaria*, Sdt. (mihi), three feet long, which came from the Levant, and was presented to the British Museum by Admiral Spratt, also in several specimens of *Reniera fibulata*, Sdt., *Esperia*, &c., and in one instance even *in the excavated chambers* of a *Cliona* in an old piece of stony coral from Cuba, where it is mixed up with the pin-like spicules of the species, which may be seen together with it in the mounted preparation.

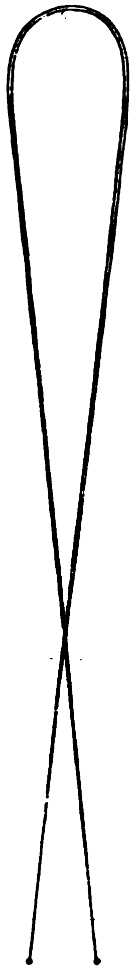
I have not yet observed it in any of the Rhaphidonemata—although, on the other hand, the Cavochalinida, ex. gr. *Tuba* (Duch. de F. et M.), which also chiefly come from the seas between the Americas, are, as before stated, commonly infested by the "isolated sunken polyp" or *Palythoa*.

Then, again, although it is prevalent in several kinds of the Psammonemata besides *Hircinia*, it seems to have almost an antipathy to the officinal sponge, in the midst of which it may be seen to polymorphose the whole of the *Hircinia* (when the two have thus grown together), without sending a single filament into the *officinal sponge.*

After these statements it need hardly be added that the filament is a parasite affecting many kinds of sponges, and that therefore it cannot form a specific character of any. It will now be described, then, as such, in the *mass* or *tissue* and in the *element* respectively, under the name proposed for it in 1871, viz.:—

Spongiophaga communis, Cart.
(See figure.)

Tissue, when fresh, soft, flexible, gelatinous; when dry, papyraceous, tough, and when torn, in this state, tomentose. Composed of fibrillæ replacing partly or entirely the sarcode of various kinds of sponges, chiefly the Hircinida. Fibril about one third of an inch long, composed of a fusiform filament terminated at each end by a bulbous inflation which is similar; filament 1½-6000th inch broad in the centre, diminishing gradually on both sides to half this diameter at the extremities; bulbous inflation more or less ovoid with the narrow end towards the filament, averaging 2 by 1⅓-6000th inch in its greatest diameters; filament consisting of a transparent sheath filled with a gelatinous colourless substance in which no structure is visible until solution of iodine in hydriodate of potass is applied, when it becomes of an amber colour, assumes a spiral form, and the whole filament, if doubled upon itself, becomes rapidly intertwisted like the strands of a rope, returning to its natural state both outwardly and inwardly when the solution of iodine is washed out with water, so as to reassume its original appearance in every way. Internal contents



Spongiophaga communis (artificially arranged).

Scale about 1-24th to 1-1800th inch.

slightly issuing from the broken ends of a divided filament, where they contrast strongly with the colourless state of the sheath under the application of the iodine solution; sheath circularly corrugated from retraction at this part, and presenting lines of corrugation on the *inner* side of a bend, but *no septa internally.* Contents of the bulb apparently the same as those of the sheath, with the addition of an indistinct nuclear body surrounded by a granular plasma, presenting a vacuole in the centre, but very variable in appearance in these respects, becoming of an amber colour under the effect of iodine, not purple like that of potato-starch &c. Filament sometimes swollen in the larger part by a nuclear body like that of the bulb, and, in like manner, often slightly acuminate at one point. When dry, highly hygrometric, twisting about on the field of the microscope on being breathed upon, like the elaters of an *Equisetum*-spore similarly circumstanced.

Hab. Marine. Infesting and destroying the sarcode of many kinds of sponges, especially the *Hirciniæ.*

Loc. Worldwide.

Obs. This parasite is not a commensalist, but a devourer of its host, like the seaweed *Thamnoclonium flabelliforme*—finally, in the Hircinida, replacing the entire sarcode so as (as before stated) to present a pseudomorph *only* of these sponges. Sometimes a few fibrillæ are a little thinner than the others in the rest of the mass; and in some sponges they are altogether thinner than in others, as in *Sarcotragus spinulosus*, Sdt., where they are *all* thinner than in *Hircinia variabilis*, Sdt., as seen in the type specimens of these sponges respectively in the British Museum; but this is the only difference that I have observed in them worth noticing in a developmental point of view. The bulb often varies slightly in shape; and the filament appears to be sometimes *once* branched; but in what form the branch terminates I am not able to state, having only observed it once; besides, these varieties can only be viewed as anomalies. Under no circumstances have I been able to satisfy myself that the contents of the filament are septate. As with the smaller, coreless, horny sponge-fibre, so with this filament, decomposition of the contents of the interior leads to the formation of oleaginous globules, which, presenting shades of colour varying from ochraceous yellow to rusty red, cause the tissue formed by them to present these colours respectively.

Although dyeing with magenta and mounting some of the filaments of a specimen that I possess which has been preserved in spirit has given the *entire* form, nothing that I have yet seen has led me to a knowledge of the mode of reproduction and development; nor have I ever noticed any more decided difference in the size of the filaments than that mentioned. The whole of this part remains for future observation to determine; and it appears to me that such information can only be obtained from *living* specimens.

The filament resembles *Vaucheria* in its contents being continuous and not septate. *Vaucheria* also presents a faint resemblance to it in the terminal enlargements of its filament, which here are for reproductive purposes; but there is no chlorophyl in *Spongiophaga communis*, and in no other respect is it like *Vaucheria*.

There is an entophytic Saprolegnious cell (? *Pythium*, Pringsheim) which bores its way through the sheath of *Spirogyra*, especially under conjugation of the latter, and, entering the sporangium by tubulation, again becomes inflated there, nourishing itself with the contents of the sporangium, and finally producing a young brood in the inner cell or inflation, which may escape into the sporangium itself—or in the outer inflation, where the embryos may escape into the water—probably in these respects being influenced by the best prospect of support. Here, of course, there is no chlorophyl, and there are no septa in the tubulation, while the contents, until they become differentiated into a new brood, appear to be composed of structureless transparent plasma, presenting throughout nothing but an amber colour on the application of iodine.

How far *Spongiophaga communis* may be allied to the Saprolegnieæ I am not able to state; while its habits so far resemble those of *Thamnoclonium flabelliforme* as to produce in some *Hirciniæ*, as before stated, a pseudomorph of the sponge, in which hardly any thing more remains than the foreign objects which formed the core or axis of the horny fibre.

Saprolegnious Mycelium.

In 1845 ('Annals,' vol. xvi. p. 405, pl. xiii. figs. 1-6) Dr. Bowerbank described a new genus of sponges under the name of "*Auliskia*," which was characterized by the presence of "minute cæcoid canals radiating from the fibre in every direction." These, however, Schmidt, in his critique on the synonyms and species of the Keratospongia (Spong. Adriat. Meeres, 1866, 2nd Suppl. p. 10), considered algoid, and therefore rightly observed that the genus should be suppressed. I had also observed it in two or three instances, and had regarded it in the same light—that is, of the same nature as the tortuous, branched, tubular filament which sooner or later infests almost every hard marine organization, both kerataceous and calcareous. How far it may occur after death I am not able to state; but it is present in the fibre of *Aplysina capensis*, Cart. MS. ('Annals,' 1875, vol. xvi. p. 192)—that is, a reddish, purple, massive hircinoid *pseudoceratinal* sponge from Algoa Bay, which, from the presence of the sarcode, appears to have been living when taken up for preservation.

Thus Dr. Bowerbank's genera respectively, viz. "*Stemmatumenia*" and "*Auliskia*" were founded on the presence of a parasite, and that following, viz. "*Cartilospongia*" (*ib.* p. 408, pl. xiv. figs. 6–8), upon the structure of a compressed, circular, cake-shaped piece of bone! Curiously enough, in examining Dr. Bowerbank's collection, the identical bone has come before me, which appears to be the body of a *fœtal* whale's vertebra, bearing the exact dimensions and descriptive characters given by Dr. Bowerbank (*l. c.*). At first sight it is very much like the skeleton of a sponge of this shape; but the odour evolved by making a vertical section of it through the short axis, and the microscopic examination, place beyond a doubt its true nature. As Dr. Bowerbank was a good observer, his description and illustrations are valuable from their correctness; but his inference was incorrect.

FOREIGN OBJECTS.

Although these, being without life before they were taken up by the sponge, cannot be considered parasites, yet there is one which so frequently occurs in the Psammonemata, so like a mineral product, and often so abundantly, that it demands a passing observation here. I allude to a little prism of *calcite* banded occasionally with yellow, brown, red, or amethystine colours, separately or more or less united in the same prism. It occurs in these sponges generally, but most plentifully in the Arenosa from Port Jackson; and hence I thought at first that it must come from some mineral source there; however, one day finding groups of these prisms *in situ* in a large specimen of an *Esperia* from Southern Australia, which had also enclosed some bivalve shells like *Crenatula phasianoptera*, I was led to compare them with the structure of the latter, and immediately saw that, everywhere, their source must be from the disintegration of thin shells like this, which are made up of similar prisms, coloured in accordance with the shells from which they are derived.

DENDRITES.

Very often, on old kerataceous fibre, little, colourless, circular dentritic spots make their appearance whose structure is so minute that even under a compound power of ¼ inch with high ocular it does not appear satisfactorily. All that can at present be stated of them is, that they are composed of branched filaments which radiate from a central point; but whether they are algoid or fungoid, or what their real nature is, future observation must determine.

ROT.

Lately several complaints have been made of the rapid washing away of officinal sponges after they have begun to be used; and on microscopical examination of such sponges before and after they have been brought into use, it would appear that while the superficial fibre is all continuous, that within is broken up into short pieces. How and when this occurs I am unable to state, further than that, like dried fish not properly cured, the surface may remain good while the interior becomes broken down by putrefaction; or it may be from some chemical substance used in preparing them for sale, which has not been thoroughly washed out from the interior; but the surface remaining sound in each instance would ensure their sale until the unfortuate purchaser finds out that, after a little usage, they become reduced to nothing, and that the soundness was merely superficial. Perhaps the best test of a sound sponge is the extent to which it expands, and *vice versâ*, after having been filled with water. Those which are broken down in the interior, not having the same amount of resiliency as the rest, will probably vary little in size by the change. (For an excellent account, with illustration, of the mode in which the officinal sponge is obtained in the Levant, see ‘Travels and Researches in Crete,’ by Captain (now Admiral) T. A. B. Spratt, R.N., C.B., F.R.S., &c., vol. i. chap. xx. p. 215, 1865: Van Voorst.)

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UNITED STATES PUBLIC HEALTH SERVICE
RUPERT BLUE, SURGEON GENERAL

BREEDING OF ANOPHELES QUADRIMACULATUS IN DEEP WATER AND AT A DISTANCE FROM SHORE

BY

H. R. CARTER
Assistant Surgeon General
United States Public Health Service

REPRINT No. 463
FROM THE
PUBLIC HEALTH REPORTS
APRIL 19, 1918
(Pages 571-572)

WASHINGTON
GOVERNMENT PRINTING OFFICE
1918

BREEDING OF ANOPHELES QUADRIMACULATUS IN DEEP WATER AND AT A DISTANCE FROM SHORE.[1]

By H. R. Carter, Assistant Surgeon General ,United States Public Health Service.

It is generally stated that Anopheles do not breed in deep water or at a distance from shore. This is usually true because (1) such water does not furnish food for the larvæ: (2) they are drowned by wave action; and (3) they are eaten by fish.

In the course of malaria surveys made by the United States Public Health Service near the United States Marine Barracks, Quantico, Va., in September, 1917, the contrary was found to be true.

Examination up to July 29 had shown no production of Anopheles in either Quantico or Chappawampsic Creek and no production of *A. quadrimaculatus* anywhere except in pools near the mouth of Creek No. 1. Since this was scanty and had not been found up to July 16, it was feared that this species would appear in Quantico and Chappawampsic Creeks as the season advanced. Being large, these creeks would warm up more slowly and hence breed later than small pools, and this species is not an early breeder.

On September 3 an examination was made of a number of houses at Quantico and *A. quadrimaculatus* were found in them in such number and distribution as to indicate an important source to the northeast of the village. In addition 442 mosquitoes were taken in an isolated group of tents one-third mile distant from the lower part of the Chappawampsic. No other water not under control was within a mile.

Examination of Quantico and Chappawampsic Creeks was made by boat by Mr. Messer of the Virginia State Board of Health and myself. In both creeks there were large masses, acres in extent, of "floatage" over the wild celery (*Vallisneria spiralis*).

This plant was growing in water from 2½ feet to over 6 feet deep and extended in places from one-quarter to possibly one-half mile from shore. Its long blades floated just level with the surface of the water rising and falling with the tide and pointing down the current. The "floatage" was formed mainly of the broken pieces of these blades—they are quite fragile—and an enormous amount of pollen of the same plant; it was then in flower. In a few places the "floatage" was bound together by a growth of alg but mainly adherent without it. In these creeks there was th heaviest breeding of *Anopheles quadrimaculatus* I have ever se over a large area. The patches of "floatage" were measurable i acres and fractions of an acre rather than in square yards. Quantico Creek they were at both sides of the channel, but no within 300 feet, say, of the shore. In Chappawampsic Creek th were mainly near the mouth and on the side next to the cam Here they were within half a mile, maybe less, of the tents in whi we found the Anopheles and were most probably their sourc Quantico Creek is about a mile wide where this "floatage" w found. Chappawampsic is about 1¾ or 2 miles wide at its mouth. W estimated the average number of larvæ per dip at 8, but one dipp took 52. They were of all sizes, and pupæ were also present. In addition to the breeding in this "floatage" there was breeding in some of the lotus beds, where the leaves of this plant fell *into* th water and adhered together as they decayed. Where the leaf stoo up above the water there was no breeding. The same condition breeding in floatage in deep water, had been noted on Broad Riv S. C., and other places in our work on Impounded Waters, but so spectacular as this.

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[1] Reprint from the Public Health Reports, vol. 33, No. 16, April 19, 1918, pp. 571-572.

In all of these cases the matting of the "floatage" prevented t breaking of the waves, which passed through it in long smo swells, and furnished a perfect protection against fish. It m also have provided a good food supply.

The problem of the control of this breeding is a very difficult o

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ter, H. R.

YELLOW FEVER INSTITUTE, BULLETIN No. 10.

Treasury Department, Bureau of Public Health and Marine-Hospital Service.

WALTER WYMAN, Surgeon-General.

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THE METHODS OF THE CONVEYANCE
OF
YELLOW FEVER INFECTION.

(Revised Edition.)

By Surgeon H. R. CARTER.

JULY, 1902.

WASHINGTON:
GOVERNMENT PRINTING OFFICE.
1902.

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C.—TRANSMISSION. **Asst. Surg.-Gen. J. H. WHITE, Chairman.**

E METHODS OF CONVEYANCE OF YELLOW FEVER INFECTION.

By Surg. H. R. CARTER.

JULY. 1902.

hether further experimentation is desirable before we accept with-
reserve the *Stegomyia fasciata* contaminated by a patient sick of
w fever as the *only* natural means of conveying yellow fever may
question, yet I think that the argument for its being in nature con-
d only by a host, and doubtless a mosquito host, is strong. It is
med that there is no question that it may be thus conveyed.

Analogy: Of such diseases as have been fully investigated, all
are conveyed from living hosts are caused by animal parasites.
of these diseases each one *is conveyed (in nature) only by means of a*
of a given genus.

Agreement of facts observed about the propagation of yellow fever
occurs in nature with the necessary deductions from the theory of
eyance by this host.

) The fact that the disease is not directly propagated from the sick
e well, that it is propagated only by means of an infected environ-
t.

) That a man sick of yellow fever may infect his environment.

) The existence of "infectible" and "noninfectible" territory. (*a*)

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n "infectible" place is one which, if cases of yellow fever be introduced therein,
mes infected—i. e., capable of inducing yellow fever in those who, susceptible to
disease, visit or live there. A "noninfectible" place is one which will not become
ted even if cases of yellow fever be introduced. In such a place yellow fever is
a transmissible disease. The theory of conveyance by a host would explain the
rence by the presence of the proper host (*Stegomyia fasciata*) in the one and its
nce in the other.

To me the mass of this evidence is sufficient to be convincing, and I count it prov that baggage from Havana and Vera Cruz will not convey yellow fever directly to pe ple after the voyage to New York or Spain, thus that yellow fever is not conveyed b mosquitoes carried in such baggage.

Whether this time element is necessary, of course, is not determined by these obs vations, but it is a factor in them.

With the above should have been considered in addition from 175,00 to 200,000 persons, troops, and families returned from Cuba by transpo at the end of the war, with baggage and equipment.

It is known that yellow fever is conveyable from the sick only b means of an infected environment. The contrary has been the teachin of the conveyance from fomites. That it can be *directly* conveyed t man from infected fomites is the doctrine which has been taught by al who have held to conveyance by fomites. All the instances reported of conveyance by fabrics, of which I have cognizance, are eithe explicitly ascribed to, or are explicable by, this direct conveyanc Now, if the "infected" fabrics can convey yellow fever *directly* to men, obviously they will convey it to susceptible people who come into direc contact with them in any place, whether the locality be "infectible" or "noninfectible." Therefore, as against the possibility of direc conveyance of yellow fever from fabrics to man, the data from Ne York and from Spain, which I have given above, if conclusive for tho places, are conclusive against such conveyance in places known to b "habitually infectible."

As to the positive instances of conveyance by baggage which have bee reported, none that I have been able to inquire into personally, certainly exclude this method of conveyance. The reporter begins (as I did) wit the assumption that yellow fever is conveyed by fomites, and as soon as h can show the introduction of fomites from an infected place, he is satisfied He looks no further for any other source of infection. Strange to say the *best* attested cases I have come across have not been those of conve ance by *packed* baggage, but *by clothing worn on the person*, sometimes f a number of miles, or hours through the sun and air, which is again all other experience of this disease. Of the older reported cases I ha nothing to say; I can not examine them; only as none of the mode ones which I have been able to inquire into clearly exclude other mea of conveyance, it is fair to at least doubt if the older reports are imp cable.

Also it is common sense, that when there is a great mass of negat testimony on a subject, all tending one way, we may not accep moderate amount of positive evidence in rebuttal unless its *data be c clusive*, and I think the evidence of the Cuban and Mexican baggag great enough for this rule to apply.

Note, too, that the argument for conveyance by fabrics is reall negative one. The reporter says, "there was no possible source of fection except the fomites." He means that he knows of no ot

Exactly the same argument was used, and logically, for the spontaneous origin of yellow fever. Cases were cited for which no exposure to *any* source of infection could be made out, and the statement was made that there *was* no exposure. From this, if admitted, the admission of the "spontaneous origin" of the case was a necessary consequence. These cases were (indeed are) not a few. They were reported with as much detail, and with as much care, and by as good men, as those for which no exposure except to fomites is known. The argument for the spontaneous origin of yellow fever, and that for its conveyance by fomites, both depend on our accepting that there were no exposures unknown to the reporter.

I think then we may reject the conveyance by fomites as not proven, and say that there is no good evidence in rebuttal of conveyance save by a host.

Now, let us look at this evidence. The argument from analogy, in this case has great weight. The presumption that it creates is strong.

For the second argument. It is indirect evidence, of course, yet it is the kind that we accept for the the theories of the causation of other kinds of natural phenomena. That the deductions from the theory agree with the observations of the phenomena is the only evidence we have for the doctrine of universal gravitation, of the vibration of light, of the conservation of energy, etc., which physicists hold as beyond cavil. It is true we have been able to apply mathematics to these theories, and that the chain of phenomena which we can thus compare is much greater than we have in this instance; yet I think that we can fairly claim that in our case *all the phenomena which we have fully examined* agree with the deductions from the theory and none disagree, and the *principle* of deciding on *the truth of the theory is the same.* I think then that the evidence (presumptive only) is good, that the only means of conveying yellow fever is by means of a mosquito host.

This evidence is indirect and presumptive only. Yet it seems easy to obtain *some* direct, although negative, evidence—i. e., to show that other methods (besides the host) do *not* convey infection. Could we show that *no* other methods *ever* convey infection it would be a demonstration.

To look at the matter broadly, there are usually three atria of entrance of an infection: (1) By the alimentary canal; (2) respiratory tract, and (3) abraided or punctured integument. For yellow fever the first is not accused. The negative evidence against conveyance by drinking water is overwhelming, and while that against foods is less so, still it is great, and there is no evidence whatever *for* it is so far as I know, and no one claims it.

To exclude the second one. If we could place a certain number of people in an environment infected with yellow fever—i. e., an environment in which cases of yellow fever *were being contracted* (not simply where such cases existed), living in all respects like those who are contracting the disease, but protected against mosquitoes, and these people should fail to contract the fever, it would be evidence that the single factor present in the case of those contracting the fever, but absent in their own—i. e., the mosquitoes puncturing the skins of one set and not of the other, was the cause of the different results in two sets of people. I say "evidence," and this evidence would be convincing in proportion to (1) the degree of infection of the premises as shown by the proportion of those entering them who contracted the disease and (2) the number of susceptible people exposed and the length of their exposure. It is somewhat strengthened if these same people who escape afterward develop fever from mosquito inoculation; but this is not necessary if enough people are subjected to the experiment. Such evidence would never, strictly speaking, be proof; yet could such an experiment be made in a house like the Grey House, at Orwood, in 1898, where of 32 people who entered the house, some only to stay a few hours, 31 developed yellow fever *from that exposure*, the exposure of a very moderate number of people, not immune to yellow fever, thus isolated from mosquitoes, would be convincing to me. I can conceive of no other test which will *directly* show that conveyance of yellow fever does not take place by the respiratory tract.

We have seen that the theory of conveyance by a host explains all the facts which we know of the conveyance of yellow fever. Does any other theory also explain them? If so, it also has a right to be considered.

It is proven that yellow fever is not in nature transmitted directly from the sick to the well. It is propagated from the sick man by his infecting his environment, from which only is it contracted by others exposed thereto. This is nowise in accord with the methods of conveyance of known bacterial diseases, all of which are conveyed by material directly from the patient inhaled or ingested by, or (as with leprosy) applied to an abraded surface of, the one who receives the infection.

From this it is a *necessary* deduction that the disease is not in nature propagated by material conveyed directly from the sick to the well; that some material after leaving the body of the patient *undergoes some change* outside of his body, and *after* this change produces yellow fever in one susceptible to that disease.

This change, which takes place outside of the body of the sick man, may be:

A. A change in physical condition of the excretion containing the microorganism rendering it able to reach the proper atrium for the infection of others. Analogous to the drying and pulverization of tubercular sputum, etc.

B. The infection of a host, by which host (or its offspring) the microorganism is transmitted analogous to the transmission of malarial fever or the Texas fever of cattle. This may involve no change in the organism itself. It may simply place the organism in a position to enter the human organism by the necessary atrium, as directly into the circulation or lymph spaces. Of course it does not negative such change.

C. A change in the microorganism itself, it undergoing development outside of the human body into some stage different from that in which it leaves the body. This last has been the theory accepted by most of the writers who have considered this subject, but there is no analogy with the propagation of any other disease, so far as is known to the writer.

No other hypothesis is to me conceivable.

Also it is proven that not all environments become infected—i. e., capable of inducing yellow fever in those exposed in them by the presence of people sick of yellow fever; that there are places which become, habitually become, infected if cases of yellow fever be introduced at certain seasons of the year and others which do not and into which the introduction of such cases causes no infection of the environment.

Certain conditions of the environment, then, even in the presence of the yellow-fever patient, are necessary for it to become infected. Besides temperature, moisture, and altitude, there are others of which we are ignorant, and many localities habitually have not received infection from the presence of the sick, although there are no differences in soil or climate, which we could appreciate, between these localities and others that did so become infected.

This quality of a place, its being infectible by yellow fever, sometimes, probably frequently, varies from year to year. Cases of yellow fever may be introduced some summers with impunity, and in others in the same place spread on introduction. Compare the results at Fernandina in 1876 and 1877.

Now, of the three hyposeses, A, B, and C, the first is, I think, not compatible with what is known of the infectibility of places, and which is so well known that we have only alluded to it above. It is not reasonable that the conditions which determine the physical condition of the infective material should be those that we know to influence the infectibility of places, or that they should be so elusive to meteorological tests as the latter have proven.

The third is negatived by the proven fact (Reed and Carroll) that the blood or serum of a yellow-fever patient injected hypodermatically into a well man will produce the disease in him. The microorganism then exists in the blood of the patient, and consequently in the bloody material extruded from him (gums, etc.) in a form capable of producing the disease. No change of development is necessary if it can only be introduced into the circulation directly.

The second hypothesis of the three, which alone seemed admissible, is then the only one not incompatible with the facts which we know of the propagation of yellow fever, and we have seen previously that it is *entirely compatible* with them.

To sum up:

For the belief that yellow is conveyed in nature *only* by a hos and doubtless a mosquito host, we have—

1. The analogy of other diseases conveyed by insect hosts.
2. That all facts observed about the propagation of yellow fever agree with the necessary deductions of this theory.
3. No other theory explains all the facts observed of its propagation.

The second postulate, may be, I think should be tested further by the collection of other facts to be compared with the theory; possibly on the lines given above, for obtaining direct negative evidence about this theory of conveyance. Yet the evidence already extant seems to the writer convincing and he is convinced that yellow fever is conveyed in nature *only* by a mosquito host of a particular kind or kinds.

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UNITED STATES PUBLIC HEALTH SERVICE
RUPERT BLUE, SURGEON GENERAL

EFFECT OF ANOPHELES PUNCTIPENNIS ON THE NATURAL CONVEYANCE OF MALARIAL FEVER

BY

H. R. CARTER
Assistant Surgeon General
United States Public Health Service

REPRINT No 464
FROM THE
PUBLIC HEALTH REPORTS
APRIL 19, 1918
(Pages 572-575)

WASHINGTON
GOVERNMENT PRINTING OFFICE
1918

EFFECT OF ANOPHELES PUNCTIPENNIS ON THE NATURAL CONVEYANCE OF MALARIAL FEVER.[1]

By H. R. Carter, Assistant Surgeon General, United States Public Health Service.

There is no question that *A. punctipennis* is capable of conveying malarial fever. King first (1915) showed that mosquitoes of this species were readily infectible and that the malaria parasite underwent complete sporogenous development in them. Mitzmain later (1916) produced malarial fever in men by the bites of infected mosquitoes of this species. To what extent they convey malaria in *nature*, however, is not determined.

It has been shown (Public Health Bulletin No. 79) that this mosquito is rarely found in residences—houses occupied by men—in the day time. Where both species are breeding to the same extent *A. quadrimaculatus* is found in such houses in far greater numbers than *A. punctipennis.* If this is because *A. punctipennis* do not enter the residence to feed, it would imply that they are less efficient as vectors of malaria than *A. quadrimaculatus.* It may be, however, that they enter residences at night, feed and leave before morning. Such evidence as we have is against this, but it is not enough to be convincing. It was purposed to determine this point last summer, but owing to the war this study could not be carried out. Indeed, observations to determine the general problem "To what extent is *A. punctipennis* a vector in nature" had been planned from several sides, biological and epidemiological, but were necessarily abandoned when the extra-cantonment work was taken up. The following epidemiological observations, however, bearing on this subject, were made:

A malaria survey was made in August 1917 of Camp Meade, Md., and its environment—about 18 square miles. Eight residences gav a history of malarial fever in 1916 and 1917 at four places. Examina ion of 74 places breeding Anopheles showed 5 of them producing *A. quadrimaculatus* or *A. crucians.* The remaining places were breeding only *A. punctipennis.* Punctipennis were also found in three o the other places with the other species.

Note that malaria was reported in houses near all the places producing *Anopheles quadrimaculatus* or *crucians* except one.[2] O some 300 houses where the malaria history was taken, the great majority of which were near places producing punctipennis, only one reported malaria. The country was, as seen from the number of houses, very thickly settled, mainly by whites, and in the part most thickly settled the breeding of Anopheles (punctipennis) was as general as I have ever seen. I don't mean as profuse, although it was free and in places profuse.

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[1] Reprint from the Public Health Reports, vol. 33, No. 16, Apr. 19, 1918, pp. 572–575.

[2] There was a house near this place, but, as it was not numbered on our working map, it probably w not visited or there was no one at home when it was visited.

It is obvious that the diagnosis of malaria based on the statements of patients can not be accepted without reserve, but at four of these houses (all near breeding places of *A. quadrimaculatus* or *A. crucians*) the history of repeated chills and fevers, given by intelligent people, was very clear, and Derivaux states that he found a number of such cases (in 1915) near the other place breeding quadrimaculatus. The statement of those claiming freedom from malaria can, I think, generally be accepted, as it involved freedom from any kind of sickness. The census of the houses was made by the State board of health, but special inquiries, at five of the houses, were made by myself.

In this locality, then, the production of *A. punctipennis* in large number and adjacent to many white people was not associated with malarial fever, while that of *A. quadrimaculatus* and *A. crucians* was.

This may be correlated with our observations at Greenville, S. C., in 1914 and 1915. In June, August, and September a stream running through the heart of this town was absolutely swarming with *A. punctipennis* larvæ and the imagos were abundant in culverts, spring inclosures, etc. Yet Greenville was practically free from malaria. There was some malaria, but it was scarcely a sanitary problem. The same was true of Rock Hill, S. C.: a large production of *A. punctipennis* and only a very moderate amount of malaria. The same appears to be true of the country around Atlanta. Grimm, of the Public Health Service, states that in a large number of blood specimens examined at Spartanburg, S. C., during two and one-half years, he failed to find the malaria parasites except in cases that had recently come from malaria sections. Yet Le Prince reports *A. punctipennis* breeding freely in this section last fall, and I think this was true in 1914 also.

During the investigation of impounded waters some country neighborhoods were found with a large number of punctipennis and only a moderate amount of malaria, contrasted with communities in which quadrimaculatus was prevalent and malaria decidedly common. For instance, a survey of the Valley of Broad River, S. C., from Dawkins to Blair, was made by Griffitts in 1916. Punctipennis was breeding freely in all of this section, which, however, was not considered as malarious (measured by the standard of this section) except at the Buckhead Settlement. Here it was prevalent, notoriously so, and here *A quadrimaculatus* was breeding freely in some large ponds. More imagoes of this species were taken in the first house examined at Buckhead than in the 12 days' work in the punctipennis district. Several somewhat similar instances could be given from these investigations. Observations consistent with the above have been made at other places.

I have never found malaria prevalent where only punctipennis was breeding, but Brumfield of Virginia has reported quite an outbreak of malaria where this was the only species. There was also a sharp epidemic at Greenville, S. C., in 1912, although whether punctipennis was the only form then present is not known.

The above report of conditions about Camp Meade is made so that other men may be encouraged to record observations of similar (or contrasting) conditions, i. e., the relation of the prevalence of *A. punctipennis* to the presence or absence of malarial fevers. The definite determination of the efficiency of this species as a vector *in nature* compared with *A. quadrimaculatus* and *A. crucians*, is of very great importance to the sanitarian. If it is not a vector of serious sanitary importance—for which there is certainly some evidence—we will avoid undertaking the very large amount of work required for the control of its production. Its breeding habits are quite different from those of *A. quadrimaculatus* (and to a lesser degree from those of *A. crucians*) both in character of place and in season, and special measures are required for the control of this species not needed for the others. Over the whole United States the cost of these and other measures for controlling punctipennis would run into many millions each year, which should not be expended if the malaria averted by them—for punctipennis unquestionably does convey some malaria—causes less loss than would the expense of averting it. To spend a dollar in sanitation and get less than one hundred cents' benefit is not only bad business, but bad sanitation.

To what extent each of the three species of Anopheles most common east of the Rocky Mountains, *A. quadrimaculatus*, *A. crucians*, and *A. punctipennis*, is a factor in the *natural* conveyance of malaria is possibly the most important of our field problems.

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BIOLOG

w, H.K.

UNITED STATES PUBLIC HEALTH SERVICE
RUPERT BLUE, SURGEON GENERAL

SCREENING AS AN ANTIMALARIAL MEASURE

7

BY

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REPRINT No. 183
FROM THE
PUBLIC HEALTH REPORTS

APRIL 17, 1914

[REVISED FEBRUARY, 1918]

WASHINGTON
GOVERNMENT PRINTING OFFICE
1918

methods of screening judged to be the best. That such a paper is advisable—nay, necessary—is evidenced by the fact that the writer has seen no single house perfectly screened against Anopheles mosquitoes since he left the Canal Zone, and he has seen and examined many.

There are certain disadvantages connected with screening, some of which are inherent and some due to faulty methods of application. Screens (1) keep out the air; (2) prevent the use of the window openings to throw things out; (3) make going through the doors more troublesome; (4) are troublesome to keep closed; and finally (5) are an item of cost.

Screening properly planned should avoid these disadvantages as much as is compatible with efficient protection against mosquitoes and proper planning will minimize them.

Openings to be Screened.

The openings to be screened are of three kinds: Verandas, doors, and windows, and screening is differently applied for the three.

For verandas the screening is stationary: For doors the screens swing on hinges like doors: For windows they are stationary, swinging on hinges, or sliding up and down, as the case may be.

Unless deterred by cost every house furnished with a veranda should have that veranda screened, or a part of it at any rate. Of course, the veranda need be screened only if it is used for a sitting room in the evening. But this is an almost universal practice in malarial districts during much of the malarial season.[1]

The screening of a veranda is costly because it takes a considerable quantity of netting. Since this netting is fastened on permanently it is exposed to weather the year round, instead of being housed during the winter, as is the case with detachable screens, and hence does not last as long. There is, however, no expense for frames and hardware as for window and door screens, and such doors and windows as open on the veranda need not be screened. On the Isthmus, where labor was high and where all screens must remain in place 12 months in the year, it was considered almost as cheap to screen the verandas as to screen the doors and windows opening on them. In the United States this would not be true and the expense of screening verandas will frequently be deterrent. Yet where it can be done the added comfort and safety—for the veranda will be used as a sitting room at night—

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[1] Observation during the year 1916 (LePrince and Griffitts) showed that *Anopheles punctipennis* frequently bites on porches in the evening. This species was not observed biting in the house, and in a large number of observations (1914 to 1917) was very rarely found inside a residence in the daytime. *A. quadrimaculatus*, on the contrary, was common in residences, generally gorged with blood. (U. S. Public Health Bulletin No. 19, p. 21. Southern Medical Journal, August, 1917, p. 642.)

will more than repay the expense. Of all screening it is the safest, as it has only one opening—the door.

It is not worth while to discuss the advisability of screening the doors and windows. All windows and all doors opening out of doors must be screened, except, of course, those opening into a screened veranda. Screen doors are far more likely to admit mosquitoes than window screens, as the former must be opened frequently while the latter should be opened rarely or not at all. Screen doors also are rather costly. On the Canal Zone, therefore, only two outer doors were allowed to a house.

The things to be considered are:

(1) The netting common to all screens.

(2) Frames necessary for doors and advisable for windows.

Springs and fastenings are needed only for doors and such window screens as swing on hinges and will be considered with them.

The netting is attached to the frame by cutting it large enough to fit in a groove which goes around the frame on the inside edge, bending at right angles and coming up flush with the surface. It is secured by a square beading pressed down in this groove, the brads securing it going in nearly at right angles to the plane of the frame. It is thus securely held, the bend of the wire being a prime factor in securing it.

In screening verandas movable frames are not needed and the broad areas of lap on the boards given to the wire covered by a batten—say 2 inches wide—render a groove unnecessary. Where netting is simply tacked across window openings no groove is possible or needed, but a narrow batten is necessary else the wire will pull out. Wire secured directly by nails will not hold.

Materials Used in Screening.

Netting. Wire netting is made of various materials. In Baltimore four different kinds are offered. All of it is obtainable in widths of from 2 feet to 4 feet, the differential between widths being 2 inches. The prices given are by the roll—100 linear feet—and vary, depending on quality and sizes of mesh. Eighteen mesh, for instance, is higher than 12 mesh of the same material and width. The prices given as follows are for 16 mesh (February, 1914):

(1) Japanned iron—i. e., painted while clean and hot—$1.50 per 100 square feet.

(2) Galvanized screening at $3 per 100 square feet, $2.50 for No. 14.

(3) Sherized iron; also called "Galvanoid," sometimes "Rustless." It has a dull finish, not bright like galvanized. Dealers state it is more lasting, $3.15 per 100 square feet, $2.65 for No. 14.

(4) Bronze; also called "composition." Costs $5.50 per 100 square feet.

(5) Copper. Same cost as bronze.

In addition, I have seen very fine netting of brass wire (on the revenue cutters in the Gulf) which would almost keep out sand flies. They were reported to be expensive but very lasting.

Remembering that three-quarters of the expense of screening windows, and more than that for screening doors, is for the frame and setting, and that the length of time the screen lasts depends on the quality of the wire, it would seem false ecomony to use the cheapest netting. For ordinary use I would advise the sherized or the galvanized nettings, although they will not last as long as either the copper or bronze. The japanned iron, however, has lasted in Baltimore since 1902—10½ years, but it was on the outside of windows and under galleries little exposed to the weather, and taken down and stowed in the winter. It was painted once every two years from the third year.

The bronze netting is excellent, stiff enough, and resisting to weather. It is said to last 50 per cent longer than the above, but I have no exact figures.

The copper ought to resist the weather almost indefinitely, but would be ill suited for doors or sliding screens for windows, as it is soft and stretches easily, and a moderate push would open its meshes.

It is to be noted that the farther south we go the shorter the life of a screen, because it remains longer in use each year. Screens should be taken down and stored in the winter. Also the life of a screen is very short on the seacoast on account of the salt in the air. For instance, at the Chandeleur Islands, plain iron screening—No. 14 painted—did not last the season through. It was replaced by ordinary mosquito bar—bobinet doubled—which lasted the whole of the next season. This, of course, may also be used ashore and will last one season. It is not necessary to double it unless, as was the case above, the bar was unusually coarse.[1]

Naturally, when the netting of a screen is worn out other netting can be stretched over the frame, so it is to some extent misleading to limit the life of a screen by that of its netting. A good frame will last 20 years. Those at the Baltimore Marine Hospital were put on in 1902, by the writer, and are still good, as good as they ever were. They are ¾ inch thick for windows and 1 inch for doors, measured dressed.

Size of Mesh.

The size of the mesh of a screen is indicated by its number—thus, No. 16 has 16 meshes to one linear inch—each being, then, less than one-sixteenth of an inch wide by the thickness of the wire. One wishes to use the largest mesh compatible with exclusion of mosquitoes so as to keep out the least air possible. Also the wire being larger with the larger mesh it is less fragile and lasts longer, hence is cheaper. No. 16 will exclude Anopheles. No. 18 was adopted on the Isthmus because we wished to exclude *Stegomyia* (*Aedes*) *calopus* also, which No. 16 will not do. Where this species exists, as in our seaport towns, No. 18 is necessary for comfort, but No. 16 is sufficient for protection against malarial fever. No. 14 is passable by some Anopheles, but if already in place—where economy is important—a coat of thin paint will bring the mesh down to about No. 16 size and also lengthen the life of the screen. It must be thin so as not to close the meshes.

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[1] Screening of different material has been exposed at New Orleans, La., for some years under natural conditions of weather to determine its durability. The experiment is not complete, however.

Whether No. 12—the most common size I found in use in North Carolina—can be made safe by two coats of paint I do not know. It would be worth trying and I think it would be reasonably safe, but would then exclude more air than No. 16. Two coats of paint on heavy wire should last a long time. The experiment as to whether No. 12 mesh with two light coats of paint would exclude Anopheles could easily be tried.

There is considerable economy in the use of No. 14 netting, painted, in place of No. 16. It is difficult to paint the latter without stopping up the meshes, while No. 14 can be painted. Paint is a perfect preservative against the weather as long as it lasts. There is screening in use in Baltimore—No. 14—which has been painted four times at biyearly intervals. It can not be painted again as the mesh is getting stopped up. The ordinary japanned iron screen, No. 14, thus treated is very economical.

Frames.

As before said, frames are not used in screening verandas. Netting may also be applied to windows without frames—simply tacked down by battens. This is not, however, the best method—as will be seen later—and for windows, frames had best be used. Frames should be heavy enough not to warp—¾ inch to 1 inch for windows and 1 inch to 1¼ inches for doors, or even 1½ inches if very large—these measures being of the frame after it is dressed. Those that are immovably secured in place need not be very heavy as they can not warp; yet a light frame is much more apt to be injured in handling—taking out and putting in—than a heavy one. In the long run heavy frames are cheapest. Obviously window screens that swing on hinges—as is sometimes necessary—are doors and must be heavier than those which slide in a fixed groove or, with a groove in themselves, slide over a fixed guide. Both of these arrangements keep them from warping.

Frames must fit tight, for mosquitoes try every point patiently and with infinite perseverance looking for entrance. Where they can not fit absolutely tight let them have broad surfaces of contact, as by the flat surfaces of two boards, as Anopheles mosquitoes do not enter by such openings.

Similarly, double screen doors swinging both ways are not allowable A gap to admit mosquitoes where the edges of the doors join is inevitable. Where a double door is needed one half should be fastened and a broad flange attached to its edge for the other to close against. Possibly metal doors with rubber strips setting out from their edges would be safe. I have never seen them tried. The battens I have recommended, and used, perfectly allow for shrinkage, but would not make up for much warping. Still, I have not found that mosquitoes came in through cracks between broad surfaces or around angles. They seem to fly into holes and struggle through, not to crawl into and through them. Indeed, the writer has had little or no trouble from screen doors warping, but much from not fitting at the edges. Von Ezdorf recommends a very simple method, by the use of canvas strips, for obviating the ill effects of both warping and shrinking. He reports it as entirely satisfactory, as I am sure it must be.

For sliding screens for the lower part of windows the groove or guide on the sides prevents warping and allows for shrinkage, and there is no difficulty in fitting closely except where the top bar of the screen frame lies flat against the bottom piece of the lower sash when the latter is raised to its fullest extent. There must be some play allowed between the screen and the lower sash, else neither can be moved. Here, however, we have two fairly broad surfaces in apposition and we have not found mosquitoes to enter, even when the opening was, say, $\frac{1}{16}$ or $\frac{1}{8}$ inch wide. Von Ezdorf's canvas would doubtless be satisfactory here even were there a larger interval.

Windows.

Just one word here on the subject of screens for windows. These must vary with circumstances, as the arrangement of the window sashes, the presence of outside shutters, whether one must open the screen, and the expense.

(1) In all cases it is preferable, and very much preferable, to have the screens immovable, and thus, of course, outside, so that not even carelessness can leave them open. If they can be opened they will at times be left open and at night. The screens should be in frames and these secured in the openings with screws, so that they can be taken down and stowed in the winter. If the upper sash lowers, these screens must extend the whole length of the window. If the upper sash is immovable a half length screen to cover the opening of the lower screen is sufficient. Neither of these allows of the use of outside shutters. If it be necessary to use outside shutters, these screen frames may be made in two parts, one above the other, united by hinges. The lower frame need not be over 12 inches or at most 18 inches high, and should fasten shut by hooks or catches. One will rarely forget to close it tight; and yet that it can be opened is a source of danger, and if one can do without outside shutters it is safest to do so.

(2) Where outside shutters must be retained and the top sash is immovable the outside screen may be retained another way by

iding up just outside the upper sash. This is better than a screen side the window because it will protect the openings at all degrees f lifting the lower sash, which the inside screen would not do.

(3) When shutters must be used and both window sashes are movble a screen covering the whole opening may be swung on hinges ke a door on the inside of the window. Its frame will have to be ade extra heavy to prevent warping or have an extra fastening to ess and keep it accurately into position when closed. For windows ordinary width only one leaf of screen should be used hinged on one de and fastening on the other. This screen has the advantage of all side screens of being protected from the weather and will last longer an outside screens. It is not fitted with springs.

(4) The half screen inside the window to cover the opening of the wer sash and running up and down on guides or in grooves is familr to everyone, as is the fact that unless pains are taken to raise the wer sash to its full height large spaces are left between this sash and e screen, and between the two sashes as well, through which mosuitoes, flies—even small birds—can come. These screens are safe only hen the lower window is raised to its full extent, or close shut—when ey are not needed. If sufficient pains are taken they are safe—but ey are not screens for careless persons.

(5) The ready-to-use adjustable screens, that are put under the wer sash and held in place by its weight are—all that I have seen—seless. Indeed, they are a disadvantage, turning the house into n Anopheles trap. They do keep out some flies.

(6) A window can be screened without using a frame by covering) the whole opening, or, when the upper sash is fast (b), the lower rt of it with netting directly applied to the window frame and held place by battens—say three-fourths inch strips. This is entirely fective and in first cost much the most economical—as one saves e cost of the frame. Eventually it is not economical, as the wire will ve to be left out in the weather the whole year. The shorter the inter—as in the far south—the less important this factor becomes. is arrangement is also decidedly inconvenient to those living in the use—washing windows, etc.

Doors.

I have spoken of how doors should be fitted so as to shut against ttens and the canvas strips recommended by von Ezdorf. In addin doors should—

(1) Open outward—always and invariably—else the mosquitoes ich we can see settled on them will be introduced into the house en they are opened.

(2) Where there is choice, the door of main entrance should face e wind which prevails in the evening during the mosquito season, mosquitoes congregate on the lee side of a house. In the Southern ast country such winds are quite constant in direction.

(3) Do not attempt to get "edge fitting" with doors. It is impossib with wood. Either the door will be too tight in wet weather and n close or leave an opening at the edges in dry. They must shut again a broad surface—a batten—at the *top, bottom, and both sides* so that, n matter whether the door shrinks or not, it will shut flat and tight; provided of course it does not warp. There is a disposition among builders to omit the bottom batten, as it interferes with sweeping, but i *must* be put in. The inner part of it may be beveled to the floor f sweeping, but the outer surface, against which the door shuts, mu be *sufficient* (three-fourths inch?) and *perpendicular*.

(4) Not only should there be a broad board for a brace about the level of one's hand in opening the door, but above this board, and below it, there should be on the inside of the door strips of wood—say 1 to 2 inches wide—to protect the wire screening from the push of the hands knees, or feet of one trying to open it from the inside. Heavy wi screening thus disposed is much better. One does not push from th outside of the door. Both of these devices strengthen the door.

(5) The free edges of doors—i. e., edges opposite the hinges— which are approached by steps from the outside should be about t middle of the steps as, to enter the door, one must open it muc wider and hence keep it open longer if he comes up opposite t middle of the door, than opposite the free edge of it. This is not prime importance, but—especially where Anopheles are numerous— decided advantage.

(6) Doors should be fitted with springs which close them certain and quickly—even if they do bang. The liquid check springs and th pneumatic check springs are utterly unsuitable as, at the last, th door just crawls shut. Springs on the hinges, if kept tight, are fair satisfactory, as is the long spiral spring which the door extends as opens, only on account of the narrowness of the space between th screen door and the true door, it works at an angle which takes muc from its effectiveness. A door fitted with this spring must be fitte with a "stop" of some kind—line or cleat—else if the wind catch it partly open it may blow it entirely around—180° from its positi when shut—when the spring will have no power to close it.

In my experience—and I have seen many—the most satisfactor spring is one that is fastened to the jamb, on the same side as th hinges, on the outside, and has an arm—14 to 18 inches long—exten ing along the middle batten of the door. Along this batten is a narro steel plate, on which runs a small wheel fixed in the arm. The arm the extension of a spring coil of some power and is by it pressed again the door—this pressure, of course, increasing as the door is opene

(7) Doors must have proper automatic fastenings, so that whe shut they are secure against wind. Stops to prevent their openin too wide are an advantage on all doors. They save the springs. The are a necessity if the long spiral spring is used. Where the scree door shows a tendency to warp, two fastenings some distance apar

ay be advisable, so as to press it firmly against its battens and hold flat when shut. To use them gives some extra trouble, but one soon comes instinctively careful about a screen door and in practice they ll be used.

(8) A vestibule with two doors is rarely necessary for Anopheles. was used on the Isthmus, with a pan of burning pyrethrum in for the single entrance of the yellow-fever ward, and I have seen stibules, with smudges of red mangrove, on the Indian River, idst swarms of Culex. They are certainly an added safeguard and ere Anopheles and money are abundant are advisable.

Other Openings.

No direct opening should be left from the outside into a screened use. This goes without saying, yet what I would inculcate is that must look for these openings, and look for them very carefully. have recently seen a large veranda very carefully screened, except at the door opened inward, with six scuppers for draining rain ter from the floor cut in the baseboard of the screen frame. I uld put my finger through them and the board was only half-inch ff. Keyholes in all out doors, screen or solid, must be plugged up. isn't sightly, but a small "wad" of newspaper is efficient for this d also for temporary closing of small accidental holes in the screens. n Ezdorf, a very careful observer, lays stress on the danger of trance by the fireplace, coming down the chimney. Of this I have d no experience. Most of my mosquito work has been done in places ere there were no fireplaces in the houses. I do not doubt that opheles do come down a chimney, or some chimneys, and, if so, e entrance must be guarded. Von Ezdorf recommends cloth cked over the fireplace opening held in place by lath battens.[1] Apparently the chimneys could be efficiently stopped and, if so, ost easily by stuffing bags of hay and newspapers tightly into the roat of the fireplace. They would have to be stuffed in so as to tightly all around, not just at random. At the quarantine station Ship Island, Dr. Burkhalter tells me the tops of unused chimneys e covered with sacking tied on. This is absolutely efficient and otects at one operation all the fireplaces connected with that chim- y. It must, however, be difficult to put in place and would have be removed and replaced if a cool spell requiring fires was followed a warm one. The flight of certain kinds of Anopheles—albimanus d tarsimaculata—on the Canal Zone, as observed by Le Prince, s high in the beginning and "as it gets dark enough to be hard to e" low—"from one to ten feet above the ground." High chimneys ay, then, be safer than low ones. I think none entered by my imneys in Louisville—a 3½-story building—where Culex were very evalent and Anopheles fairly so.

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[1] Public Health Reports, Feb. 27, 1914, p. 506.

Now there is one thing about small holes by which mosquitoes may enter a screened house which we must notice. Anopheles is not a house mosquito. She lives out doors and enters a house only to feed. She enters the house at night and when it begins to get light she tries to leave it. She will try to enter all night long—possibly attracted by the scent of people, but I do not know—and will find a hole, no matter how small, if it exist. She has only a short time to get out, however, and unless the way out is fairly clear she can not do so and has to remain in the house, hiding in dark places and corners; under the bed especially. A screened house with a reasonable number of small holes becomes, then, not a protection but a mosquito trap for collecting Anopheles. This I have seen time and again. Frequently very full fed mosquitoes remain in the place of feeding even when there is a clear way out, hiding in dark places.

Mosquito Bars for Beds.

(1) *They should be of fine bobinet.*

(2) *They should not be made with a slit in the side.* Such a thing is an abomination—and it is common.

(3) *They should be hung on a frame,* and a square frame is better than a round one, over the bed. Not too high to prevent one from killing any mosquitoes which have gained entrance. By lashing sticks vertically to the four corners of the bed and joining their tops with cord a frame of cord can be made.

(4) *They should not go outside the bedstead.* Neither the headboard nor the foot should be included, but they should come inside of both so as to be tucked under the mattress all around and they should be full enough and long enough to enable one to do this easily.

(5) *They should be thus tucked under the mattress* when one goes to bed and not allowed to trail on the floor. This will allow any mosquitoes which may be under the bed to get at the sleeper. The wind too may blow the loose bar aside and lift it from the floor.

Even when the bar is perfect and perfectly arranged it does not protect when any part of the sleeper's body comes against it—as the mosquitoes can bite through the bar even if they can not come through it. This was especially noticeable with the narrow cots and hospital beds in the Canal Zone. Of course it furnishes no protection until one has gone to bed—although this being the time of greatest risk of malarial infection is the time when protection is most needed.

Good bars well arranged and carefully used furnish much protection and on account of their cheapness should be universally used in malarial countries. They, however, give far less protection than screens. *Under ordinary conditions of malaria as we have them in the United States careful people in a properly screened house are safe.*

DO NOT DESTROY—

When you have no further use for this pamphlet give it to someone else.

ter, H. R.
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UNITED STATES PUBLIC HEALTH SERVICE
RUPERT BLUE, SURGEON GENERAL

YELLOW FEVER

8

ITS EPIDEMIOLOGY, PREVENTION, AND CONTROL

(LECTURES DELIVERED AT THE UNITED STATES
PUBLIC HEALTH SERVICE SCHOOL OF INSTRUCTION BY H. R. CARTER, SENIOR SURGEON
UNITED STATES PUBLIC HEALTH SERVICE)

Lecture No. 1 (March 26, 1914)
Lecture No. 2 (March 27, 1914)

SUPPLEMENT No. 19
TO THE
PUBLIC HEALTH REPORTS

SEPTEMBER 11, 1914

WASHINGTON
GOVERNMENT PRINTING OFFICE
1914

C

YELLOW FEVER.

LECTURE NO. 1.

(March 26, 1914.)

I am directed to tell you something about yellow fever. In the ime allotted it is impossible to cover the subject unless so supercially as to be useless to you. I am therefore going to take only one spect of this disease, the one of most importance and of most interest o you.

You are, or ought to be, sanitarians, and it is the sanitary aspect of ellow fever only that I will present to you. I will not discuss the isease, as it concerns the practitioner. I am also going to assume a onsiderable knowledge of yellow fever in this class, because you ave it. I am not going over a lot of facts well known about it, not ecause they are unimportant, but because you know them, and it ould be a waste of time.

As a preliminary to the sanitation of any disease we must know ts epidemiology, and to know this satisfactorily a knowledge of the ethod of its conveyance is necessary. Yellow fever, as you know, s one of the host-borne diseases. The history of the discovery and he demonstration of this by the Army board of which Maj. Reed was hairman are known to you. The subject is extremely interesting, ut I have not time to go into it. The findings of the board were, riefly: *Yellow fever is contracted by man from the bite of a mosuito, itself infected by having previously bitten a man sick with that isease, and is only thus contracted.* The first part is a direct statement of observed facts, the latter a deduction (and a negative one) rom the facts; both are not only universally admitted but abunantly proven, which is by no means the same thing. Upon this docrine all sanitary measures for the control of yellow fever rest.

There are evidently three factors considered in the creed we have entioned above—the sick man, the mosquito, the well man. There s also implied the infective microorganism of which as yet we know ut little.

(1) You will find it very generally stated that the man is inective to the mosquito only during the first three days of his illness. his is probably true, yet I would like to give you the observations n which the statement rests.

Undoubtedly, all of the experimental cases were produced mosquitoes which had bitten men during the first three days illness. The reason was that the mosquito which infected the fi experimental case was thus infected, and as the first experiment were trying for positive results the conditions of the first success experiments were copied. Other experiments followed them. O a very few attempts then have been made to infect mosquitoes fr men sick over three days. These few have failed. It is fair to s that a certain number of mosquitoes also failed to receive infecti from man during the first three days. This would leave the mat in doubt.

The Pasteur commission, which studied yellow fever at Rio Janeiro (Simon, Marchoux, and Salembeni), however, injected fo men with the blood from yellow-fever patients in the fourth d of the disease with negative results. They had previously fou this to be a very certain method of inducing the disease, more than by mosquitoes. One of the men subjected to this experim later developed yellow fever from the bites of infected mosquito He then was susceptible to yellow fever. The commission regard the others as susceptible also, but this was not proven.

Whether these four experiments, one of which only was on- man proven to be susceptible, can be accepted as proof that the blo of no man sick over three days is infective to mosquitoes, may questioned. They are enough to render it probable, and we should safe in saying that the blood is not infective after the first few days three or four. At any rate I have given you the evidence as I rec it and you can judge for yourselves. I have not seen the report the commission since it came out in 1903 or 1904, but I believe I ha quoted it correctly.

(2) The only mosquito which has been proven to convey yell fever is the *Aëdes calopus*, which was first known as *Stegomyia f* *ciata*, and then *Stegomyia calopus*. The cause of the change name I do not know, but the Bureau of Entomology, which deci such matters, has so decided.

It is fair to say that not many experiments have been made wi other mosquitoes. Only a few Culex, I think, have been tried a have proven negative. We can not, of course, accuse such m quitoes as are found in any quantity where yellow fever does n spread. From analogy, not always a safe guide, we would susp that some other species of this genus, or maybe subfamily, wou convey yellow fever, and that none of any other subfamily would.

This is no mere matter of academical interest, but of practi importance. In Hawaii, for instance, a form known there as *Steg* *myia scutellaris* exists in considerable numbers. If, as I suppo it be of the same genus as the one which conveys yellow fever, whi used to be known as *Stegomyia calopus*, it is under suspicion as a breeder of yellow fever. On account of the position of these islands they will be exposed to the introduction of yellow fever when the Panama Canal opens, and mosquitoes which convey the disease must be eliminated. The breeding habits of the Scutellaris are not quite the same as those of the Calopus, and considerable extra expense is necessary if it also is to be eliminated. A British commission is about to study, or is now studying, the transmission of yellow fever by the scutellaris.

From the time the mosquito receives the infection from the sick man not less than 12 days must elapse before she becomes infective; that is, capable of conveying her infection to others. This period, of "extrinsic incubation" it was called when first discovered, is generally over 12 days, usually over 2 weeks, and varies with the temperature, being longer as the temperature falls.

It is somewhat strange that the only case in which this period has been found to be as short as 12 days was in the first case of experimentally induced yellow fever, that of Carroll.

When once rendered infective the mosquito remains so apparently all of her active life; in one of Reed's cases, 59 days after receiving infection.

Does she transmit this infection to her progeny? You know some hosts do. The cattle tick of Texas fever is a notable example. I think this is not true of yellow fever. It is true that Marchoux, of the Pasteur commission, working in Rio de Janeiro, reported such a case, but other attempts to do so have failed. Guiteras kept mosquitoes, the progeny of those that had transmitted yellow fever during the winter, and fed them from time to time on blood, some of the people furnishing it being susceptible to yellow fever. Rosenau, working at New Orleans and, I think, at Vera Cruz, also attempted to convey it from the progeny of infected insects and failed.

Naturally one positive experiment will outweigh a number of negative ones, but there are some things about the one observation of Marchoux that make one feel doubtful. "A light but undoubted case of yellow fever" he reported. It is sometimes not easy to be sure of one's diagnosis in light cases of yellow fever, and I wish he had not said "undoubted." I would not have used that word unless I had doubted it.

(3) The man who receives the infection. There is very little to say of him. The period of incubation in him is pretty accurately from three to six days. In 24 experimental cases and in 12 others where there was only a single exposure, the shortest time (one case only) was two hours less than three days. The longest time (one case only) was two hours more than six days. A number of cases

have been reported with shorter and longer, much longer, periods of incubation following the injection of blood or blood serum treated in various ways. This is not, however, the natural method of infection, and I think for practical work we should accept the period of incubation determined by infection received in the natural manner—by the mosquito. I need not tell you that the period of infection in man is of very great importance in the sanitary measures against yellow fever, especially those to prevent the introduction from one place to another, i. e., quarantine.

In all of this I have said no word of what is conveyed: What the mosquito takes from the sick man; what she imparts to the man who is susceptible to yellow fever; what produces the disease in him. And for good reason: I do not know what it is. Of course, since the disease is communicable, it is capable of self-multiplication, and hence caused by a living organism.

From the analogy with other diseases conveyed in similar manner the organism should be an animal parasite, a protozoan. It is a filterable virus passing through the Chamberland bougie. It is very fragile; heating to 55° C. destroys it. The blood remains infective a few days (five, I believe) under oil of vaseline, but soon loses this property if exposed to air. Many of us have attempted to cultivate this organism in various kinds of media without success; to find it in smears of blood; in sections of mosquitoes known to contain it; with various stains and differential stains; with dark field illumination; with ultra-microscopic apparatus. We tried in the laboratory on the Isthmus, but without success. Recently, about 16 months ago that is, Seidelin reported finding certain bodies, some in the red corpuscles and some in the blood serum, which he claimed to be the organisms causing yellow fever. I hope this is true, but so far his work has not been confirmed by others.

The mosquito.—Let us consider the mosquito that conveys this disease a little more in detail. Its name, genus, and species I have given you. It is a small, quick, very graceful mosquito; the day mosquito of the South. No one can mistake it. Once seen, both its appearance and feeding habits are distinctive. She is gray, and flies, as we say, "all adrift," with her legs spread out. Does not fly fast like the anopheline, but is quick in changing the direction of flight. She hovers before she bites, and as she bites, sings in a very annoying manner, and her bite is very irritating. She feeds in the daytime. I do not think the hour of the day has as much to do with it as the intensity of the light. On a dark day she will bite at noon, but generally bites from 3 or 4 o'clock to dusk and in early morning. She will bite, occasionally at least, in the night, but I do not think that she normally bites in the dark. It is a little hard to determine this by observation, and experiments, I think, have not been made.

How do you reconcile this with the belief that is so current in he South that one does not contract yellow fever in the daytime?

do not reconcile it. What I have said of her time of biting is true. do not know that the other is. I do not believe it is. I have seen great many places, small towns and places, holding "daylight ommunication" with places infected with yellow fever and beeving themselves safe in so doing, but I have never yet failed to e these places receive infection if this was kept up long enough. t was always explained one way or the other. Some one had sneaked nto the infected town at night, or had stayed after sundown, or ome clothes had been brought from it, or something; but the fact emains that they received infection. It is claimed that people living in Petropolis are able to stay in Rio de Janeiro all day without ontracting yellow fever, so long as they come out and sleep at etropolis. It may be. I have no direct knowledge of the matter. he Pasteur commission, which accepts this, makes the hypothesis hat while normal mosquitoes feed during the day, the infected ones ust feed only between dusk and daylight. This is a deduction, owever, and we have no observations to show it. In experimental ork the infective mosquito feeds just as the uninfective ones do. his may not be conclusive, as in captivity they can not choose he time of getting blood, but I can cite one instance where those ot in captivity did the same. In April, 1905, there was very little ellow fever in Panama. We had been nearly a month without a eport of a case. In some way the executive building, where many lerks worked but which was not used for sleeping quarters, became nfected, and we had, I think it was, 22 cases of fever contracted in his building within about 7 to 10 days. Now, this building was not pen until 8 a. m. and closed at 5 p. m., and while some of the men ay have worked after hours, none slept there, and the majority of hose who developed fever kept the hours above given. They were nquestionably infected—that is, bitten by infected mosquitoes—during the day between 8 and 5.

This mosquito is domestic in her habits. She is found only about he habitations of man. She is not found in the marshes or the oods or fields. In the beginning of 1903 Manson, speaking of the ntroduction of yellow fever into the Far East by means of the roposed interoceanic canal, recommended that if such a canal were onstructed it be deforested for a mile on either side to prevent the ellow-fever mosquitoes breeding there. In an article that I read efore the public-health association that same fall the ground was aken that deforestation would be useless; that if protection from tegomyia were needed depopulation of the canal banks would be ar more efficient.

Breeding places.—By preference this mosquito is a "pot breeder." She lays her eggs in collections of clear—not necessarily clean—water in artificial containers, cisterns, tin cans, etc. I said "by preference." We find her breeding in other places, but generally where artificial containers are not available. I presume that when the necessity of ovi-position occurs she will deposit her eggs in the places of election if they are available, but if they are not available she will deposit them where she can. Therefore we occasionally have them in street gutters, and in pools in the earth. Probably a pool with clear water not disturbed, with firm earth sides, might do as well as an artificial container, but certainly she does not like muddy water.

Distance of flight.—It is a small, light mosquito and does not fly far from where she is brought to life. No direct experiments have been made, but the conveyance of yellow fever from an infected house is an indirect experiment, and I think it is fair to say that yellow fever will rarely be conveyed by mosquitoes 100 yards. White would put the limit far below this, but I believe that it is quite frequently conveyed by mosquitoes across the street or to a house back of the infected one. There are two cases, one reported by Melier and one by myself, in which it seems to have been conveyed much farther; in the first, 225 meters; in the second, 152 yards. Possibly we are both mistaken and there were some other means of infection than those we accused. Seventy-five yards would be about as far as you would expect it to be conveyed. This matter comes up in maritime quarantine as to how far you must lay ships off from an infected wharf. Quoting Goldberger I would say that "on account of its flight being only by day the direction of the wind during the day only need be considered in estimating this factor, if it be much of a factor, in their flight." Wind, I need not tell you, is generally onshore during the day—sea breeze.

Distribution.—It is a tropical or subtropical species, in the United States not found north of Norfolk or Virginia Beach. Some years ago I attempted to determine their presence in Baltimore by putting out breeding cans in a great many parts of the city and found this mosquito only at one point, the fruit wharf, where vessels from the banana ports came in. The old quarantine division making the southern boundary of Maryland the division between "infectible" and "noninfectible territory" seems to be justified.

In the tropical and subtropical belt it is found in practically all of the hot, wet countries; at least on the seacoast. This is doubtless due to its ready conveyance by vessels. Fortunately, although the mosquito is distributed so very widely, the parasite, the carrying of which makes it so dangerous, has by no means had so wide a distribution.

Conveyance.—It is conveyed by vessels almost any distance. The old sailing vessels were really hosts to it, as this mosquito bred in their water tanks without limit as to either time or distance. Steam vessels carry it much less, as they have far fewer collections of water accessible to mosquitoes aboard. They probably rarely breed on such vessels, but may travel as mosquito imagos for many days.

Are they conveyed in railroad coaches? White, who has studied the subject, thinks not. They were reported to me as found only in a single instance (Evansville, Ind.) in Pullman cars in 1903. But the fact of the development of yellow fever at Bristol, Tenn., at Louisville, and at St. Louis in 1878 among railroad men employed at these stations and who contracted it about these stations, I think, can be explained only by the fact of these mosquitoes being carried by railroad coaches. Occasionally I would say, then, that they are, but that is not common.

They live a long time; one in captivity—an infected one—was kept by Guiteras five months.

Hibernation.—How do they live during the winter? Their eggs are quite frequently laid above water, and these have been known to retain their vitality for over two months and a half. Possibly this may be a method. The imagos unquestionably hibernate in the Gulf States, and may do so farther north. Guiteras kept a number without food or water in a refrigerator for three months, when they were eaten by ants. There is no question that the species hibernates as far north as Norfolk and that the imagos hibernate on the Gulf coast. I have seen them in February and March.

Do the parasites hibernate in their hosts? I think not. I have done a good deal of postepidemic disinfecting and have advocated it, but I think I was wrong; that the work was useless at least in the United States.

It is claimed that two, possibly three, epidemics in New Orleans arose from infection left the previous year, but it is certain that none has so started in Mobile, nor in the little towns along the Gulf coast, in Pensacola, nor in any of the towns of the Atlantic coast from Jacksonville to Norfolk, and generally it has not been the case with New Orleans. Now numbers of these mosquitoes must live through the winter and numbers of them must have been infected when hibernation was begun; if then they do not convey infection the next year, the parasites in them must have died. It was on this account I said that once infective the mosquito remained so during her *active* life. Infection apparently dies out during hibernation. Possibly infected females may not hibernate. The general experience of yellow fever not recrudescing the year after an epidemic is proof that generally the parasites die. The negative evidence is so strong that it seems fair to believe that the fever alleged to have been "kept over" in New Orleans was an introduction early in the season, the source of which was not known. It is a matter that could be easily settled by laboratory experiment, and, if I am correct, with no danger to the men subjected to it.

The Sanitation of Yellow Fever.

I think I have said enough to enable you to appreciate the sanitary measures to be taken against yellow fever.

First. To prevent its introduction into places where none exists. If there be no *Aëdes* (*Stegomyia*) *calopus* in the places to be protected they need no protection, as it is "uninfectible territory" in which yellow fever is not a communicable disease. You may introduce a hundred cases in the absence of the mosquito and there would never be one hundred and one. This is true of the ports of the United States from Baltimore north. It is the meaning of the old division line between "infectible" and "noninfectible" ports.

Of course the same thing holds when the mosquitoes are inactive; that is, even at southern ports during the winter. Cases of yellow fever introduced then have nothing to be infected by them. Infected mosquitoes even would not be able to leave the vessels. Quarantine is properly modified then by the condition of the port to be protected as much as the port to be quarantined against, a thing which more than once has been overlooked. And here let me warn you against unnecessary measures of sanitation. They bring reproach upon the whole subject, and being indefensible, render it less easy to defend the necessary measures; moreover, there is only a certain amount of money, time, and energy available for sanitary measures, and as in old times the tithing of mint, anise, and cummin leads to the neglect of the weightier matters of the law. It is very important, then, to take no unnecessary sanitary measures.

Second. To eliminate yellow fever where it exists. I say eliminate, not control. The latter is allowable for malaria, but I think that for yellow fever the sanitarian should be satisfied with nothing short of elimination. It is easy.

Since two hosts, one human and one insect, are necessary for the propagation of yellow fever it is obvious that a perfect control of either is sufficient for the elimination of this disease from a community. I mean that if every person developing yellow fever was immediately isolated from these Aëdes mosquitoes (that is, if no access to him by mosquitoes was allowed while he was in an infective stage) yellow fever would inevitably cease. Similarly, if there was no access to man (I mean man susceptible to yellow fever) by any mosquito in the infective stage, the same thing would occur. Theoretically, then, we can eliminate yellow fever by control of either the human or the insect end of the chain.

t which end of the chain shall we begin? Shall both ends be d at the same time? One method is not incompatible with the r.

t first sight it seems most feasible to try to control the human t, because, (1) we can tell more or less when he develops yellow er; (2) when found we can isolate him by wire screening or other- e from mosquitoes; (3) *the period of infectivity of the human to mosquitoes is very short*, three or four days at most, *during ch time only* he need be guarded.

n the other hand, these mosquitoes once infective remain so finitely, and, except as their environment creates a presumption they have or have not had access to a man in the infective e of yellow fever, we have no means of telling infective mos- oes from any other, and the only practicable means of control he insect host is by its extermination—more or less complete. he destruction of the *Aëdes calopus* mosquitoes, which have had ss to a man in the infective stage of yellow fever, as distinguished the general destruction of these mosquitoes, requires such a wledge, and hence control, of the human host as to place this ure in the class of those determined by the control of the human er than in that of those determined by the control of the insect

seems easier, then, and more natural to attempt to control the an host; and the first attempt to exterminate yellow fever after method of propagation was known, which was so brilliantly ed out by Gorgas, at Habana, in 1901, rested mainly on this od. The attempt to lessen the number of mosquitoes by de- ying their breeding places was also made, and made vigorously, he principal dependence was placed on the isolation of the in- d men, and the destruction of the mosquitoes already infected hem; the destruction of *Aëdes calopus* in general being regarded adjuvant to the main work.

e same methods were used by Liceaga, for Vera Cruz; by aldo Cruz, for Rio and Para; by White, for New Orleans; and ly by Gorgas, for Panama and Colon. Its general adoption, , shows this to be the most natural method.

l of these efforts were successful, or comparatively successful, in Habana and on the Isthmus especially, and that in Habana iantly so. Yet Col. Gorgas believes and asserts that his success abana was due to the war waged on the mosquito, directly under- n as an adjuvant to the isolation of the sick, rather than to that tion itself; to the control of the insect rather than to the control e human host. This certainly was the case in the Isthmian work. ontrolled the fever by the isolation of sick men, and greatly ned its spread, and would bring it down to the very few cases per week or even per month. But it seemed that it would not disappear, no matter how closely this work was done, and I can not assure you that it was done very closely. I do not know of any higher praise that I can give Col. Gorgas as a tactician than to tell you how he pressed home the flank attack, so to speak, when we found that the main effort was not going to be successful.

The reason that the method by control of the human host failed was because under the conditions that existed it was impossible to carry it out. We could not control the human host to the degree necessary for success. No one who has not conducted a campaign against yellow fever in a place where it is endemic, where the inhabitants do not fear it, can imagine the difficulties of finding the cases of yellow fever early in the disease, or at all; even the cases which are plain. For the light cases let me say this: An analysis of the yellow-fever statistics of Habana for the 10 years preceding 1895, published by me in 1900 in the United States Public Health Reports, led me to state that but a small proportion of the cases of yellow fever in Spanish adults in Habana, about one-eighth, I think, had been diagnosticated. Taking this into account, in addition to the cases in native children, which are practically never diagnosticated, I think the most optimistic will admit that in such places control of the human host to a sufficient degree to eliminate yellow fever is impossible—not difficult, but impossible. Our only hope, then, is to control the insect host, to destroy the *Aëdes calopus* mosquito.

This is true for towns where yellow fever is endemic. In towns where it is not endemic, as in the United States, the conditions are different. With the cooperation of the people it is or may be possible to control the human host sufficiently in such a town, especially if the town be small, or, what is nearly the same thing, if the fever be confined to a limited area in a large one. This was, I think, an important factor in the success of White's campaign in New Orleans. Although the elimination of the breeding places of these mosquitoes was pressed most vigorously the results were too quick, I think, to be due to their extermination by this method. Certainly the early control—I do not say elimination—of the fever was due to this. This was the method used successfully in the suppression of an epidemic in the little town of McHenry, in 1898, I believe, and in Hampton, Va., the next year by White and von Ezdorf. The method of conveyance of yellow fever was not known at that time and the attempts were naturally made entirely from the human end of the chain. They were successful.

In towns in the United States, then, when the people will cooperate, attempts to control the human host must always be made, for they will control the fever even if they do not eliminate it; they rill smother it; lessen its rapidity of spread so that we can tide over he time until frost with a minimum number of cases. We have no rost to help us where the fever is endemic. This was the case in the ittle town of Franklin, La., in 1897. The fever was handled by solation and fumigation with sulphur of the houses in which it ocurred. It spread very slowly and we had only 10 or a dozen cases rom September 1 until frost, which came on the night of Novemer 18—plenty of time for a full sized epidemic, but for the measures ken.

Since the essential part of this method is the discovery of the man s soon as he is sick, the cooperation of the town people is necessary. ight cases are far more readily found in such towns than where e disease is endemic because, as a rule, the whole population is sceptible to yellow fever, and a light case of one member of a mily is very apt to be associated with clearly marked cases of me others in the same house and thus lead to discovery or at least spicion.

On the other hand were the problem to rid of yellow fever a town the Tropics in which it is endemic, as, say, Guayaquil, I would ncentrate all my energy in ridding the town of *Aëdes calopus;* I ould not isolate those sick of yellow fever; I would not isolate a an; I would not fumigate a room save possibly as a protection to ose in the immediate neighborhood. As a measure of general sanition helping to rid the town of yellow fever its value would not y for the trouble it causes if indeed it had any value at all for is purpose.

The conditions of the two classes of places are different and the eans advisable for the riddance of yellow fever are also different. or the last, control of the insect host alone is to be aimed at; for e first, control of the human host should also be vigorously pressed.

One does not have to eliminate all the *Aëdes calopus* to get rid of llow fever; if they are brought to below a certain number in a wn, the disease will spontaneously die out. This is common sense ther than observation, and the number necessary to continue it ould vary directly with the ratio of people immune to yellow fever those susceptible to it. Thus, suppose 10 cases of yellow fever ere introduced into a town all of whose inhabitants were susceptible the disease; suppose, then, that there were such a number of these osquitoes in the town as were able to infect only 9 men from these sick ones, obviously the fever would gradually die out. If there re enough to infect 11 men, it would increase. If the number was ch that they were just able to produce 10 infections, the fever ould continue without increasing or diminishing. Now, if one-half the people in the town were immune to yellow fever, obviously only 5 infections would be caused by this same number of mosquitoes—one-half of their bites being wasted on those immune to yellow fever—and there would have to be twice as many to be able just to continue the fever. Obviously, then, the "critical number" of Stegomyia (as Gorgas calls the number just less than which will allow yellow fever to spread in a town), this "critical number," I say, is less in any town as the number of those immune to yellow fever decrease, and a much greater mosquito reduction is required to eliminate yellow fever from a town in the United States, where practically all are susceptible to yellow fever, than from one where it is endemic. Therefore, it is far more difficult, the work must be carried further, to eliminate yellow fever in a town of the United States than in one where it is endemic. I had rather tackle three towns like Guayaquil than one like New Orleans in 1905.

There is one other thing that is not yet in the books and yet that is fairly well known by sanitarians and which you should know. It is the spontaneous disappearance of yellow fever in towns in the Tropics. There are quite a number of towns of this kind from which yellow fever has disappeared where no sanitary work has been done and which are still breeding Aëdes, or before sanitary work was done. Corinto was unquestionably infected with yellow fever in 1905. We had an inspector there until 1910 or 1911. No sanitary work was done and *Aëdes calopus* was breeding all of this time, yet in 1911 or 1912 we landed some 5,000 marines, who made the place the base of operations and stayed there some time. No fever appeared among them. Evidently the town was not infected. Bocas is free and no sanitation was done until after the fever was gone. I think the same thing happened in some of the towns in the interior of Cuba, as Puerto Principe and Villa Clara and even Cienfuegos. It has been a not uncommon phenomenon in the Tropics. Yet most of these towns—I except the Cuban towns—are still breeding *Aëdes calopus.* How does this happen? Unquestionably by the elimination of the parasite, by the failure of the human hosts in sufficient number to continue its propagation. If all the people in a endemic center become immune and no nonimmunes come in, ther is nothing to keep up the human chain, and when the infected mosquitoes die out, if they have not bitten some one who can continu the propagation, yellow fever necessarily stops. It is a spontaneou elimination of the parasite for the lack of a human host. This do not occur in large towns. Too many immigrants come in from plac where yellow fever is not endemic and the infection is thus kept up There are also a larger number of babies born, who, of course, a newcomers and susceptible to the disease. I will say this, howeve that it would require a decidedly larger number of babies per annu to continue the propagation of yellow fever than it would of su ceptible people from the outside. When yellow fever is getting rare in a town a baby remaining in its own home would be far less exposed to it than an adult going from place to place in the town. A baby while sick would be in the house of its immune parents and would thus have less chance to spread it than an adult who would contract the disease in one part of the town and develop it in another. On both counts, then, a baby is less apt to propagate it than a susceptible adult.

One thing is also to be noted, that towns of this kind are liable to little epidemics among the natives from time to time when yellow fever is introduced. This is due, of course, to the accumulation of a considerable number of people susceptible to the fever; that is, to those born since the town was free from infection. There was one at Buenaventura in 1904.

LECTURE NO. 2.

(March 27, 1914.)

GENTLEMEN: I told you yesterday something of the epidemiology of yellow fever—enough to understand the reason for the sanitary measures to be taken to control it. I also discussed the class, the general nature, of these measures. I thought that to-day I would take up the sanitary measures somewhat in detail, not simply giving you the principles upon which they are based nor the general direction in which they should go, but the details of sanitary measures.

There are two lines along which efforts to eliminate yellow fever from a place may be made. One is by the control of the human host and the other is by the control of the mosquito. There are good reasons why in a large place where yellow fever is endemic the attempt to eliminate it by the control of the human host is futile.

I should like to tell you what was done in Panama. I do not know that anything could give you a better illustration of sanitary tactics than what occurred when we found the attempt to control the human host had failed and the attack against the mosquito was pressed to success. I want to take up these two lines now in some detail.

What measures do you take to eliminate the insect host or, I will say, to control the insect host? Here we encounter the same difficulty that we find when we attempt to eliminate other things that breed very fast, such as rats, mice, flies, fleas, etc. There is no use in attempting to destroy the individual. This does only temporary good, and only a little good after all. You will never be able thus to get rid of them. The only chance of success is in destroying their breeding places, their shelters, their food supplies, or of introducing such natural enemies as may be able to keep them under control.

This mosquito's food supply is man—ourselves; their shelters are our houses. No natural enemies have been found which will control them. We must necessarily limit our campaign to an attack on their breeding places. Fortunately this is easy. In attacking their breeding places we have only to get rid of water in artificial containers or to prevent access of mosquitoes thereto. There is no problem here of draining measures, draining streams and swamps and cleaning out ditches, as we have to do in antimalarial work. If we eliminate the artificial containers about the houses of man we practically get rid of the yellow-fever mosquito and consequently of yellow fever itself. I may say that they were reported breeding in the street ditches in

New Orleans on the outskirts of the city in 8 per cent of the ditches. This certainly was not so, however, in Habana or in Panama; and I m informed by Richardson and White that the determination of the species found in the ditches in New Orleans was made almost xclusively from the larvæ, by whom I do not know, and it is very asy to make a mistake between the larva of the Culex and the *Aëdes alopus.* At any rate, in Panama, Colon, and Habana we did not ind *Aëdes calopus* breeding in street ditches as a sanitary problem. t was entirely a question of artificial containers.

Of course, if there is a mudhole or a ditch with firm sides contain-ng clear water, I do not know but that it might not do as well as an rtificial container and that these mosquitoes might not breed there ust as well as in a jar. But the fact remains that we rarely, if ever, nd this mosquito in puddles or ditches.

You must not leave artificial water containers anywhere about the remises. Cisterns and water barrels are the main breeding places, ut by no means all. At Colon there was a very excellent officer as perintendent of the hospital. I was over one day to see a case of llow fever and while there I thought I would look around to see hether there were any breeding places for mosquitoes. He laughed d said there were none. "I knew you were coming," he said, "and eaned up everything." I did not find anything except about a pint d a half of water in a grindstone basin, and if there was one there ere a million larvæ in it. He had not thought of that. In the ecutive building in Panama mosquitoes were found breeding in the shes where the brushes for the copy books were kept. In a little fice where there were three or four desks in Gen. Wood's head-arters in Habana they were found breeding in fire pails, and in me houses rather carefully inspected they were found breeding in e little saucers in which table legs were set to keep the ants off the ble. There is not any collection of water too small for these squitoes to breed there and in numbers. We found them in the ly-water basins in the cathedral and experienced a great deal of uble in getting rid of them.

Another thing: The gutters of buildings had to be looked after tty closely. If they were crushed or bent they held water; if they re stopped up with leaves or trash they held water. I thought ere there was just a slight sagging, a bit holding a few ounces of ter, with a pouring rain averaging twice a day, there would be very tle risk of *Aëdes calopus* breeding there; but the last mosquito rk in Panama was done to remedy just that condition in the tters.

I found a child with yellow fever at the Hotel Italia and I found *des calopus* in the room where the child was sick. Where did they come from? I naturally thought of the cistern as not being screened. I went out and examined it and found it well screened. It was tight. But we opened the cistern and one or two of these mosquitoes flew out. I got down in the cistern (it was an underground affair in the kitchen) and found a few larvæ on the surface, but there were many pupæ skins scattered about. I was puzzled to know how the mosquitoes got into the cistern, which was, as I said, tightly screened. Then it flashed on me all at once that while the screen through which the water poured into the cistern at the intake was fine enough to screen out the mosquitoes; it was not fine enough to prevent the eggs and the very young larvæ from the gutters from washing down into the cistern. In that way they would develop just as well and come out of the cistern as they would if they had been laid directly in the cistern water. To correct the condition we did two things: We set the tops of the cisterns on permanently with cement and put in pumps, so that if mosquitoes developed in them they could not get out, and later discontinued cisterns altogether.

That is a thing you will have to watch for if you have any work to do in the extermination of mosquitoes. You had better not have gutters on the houses, but if you have gutters let them, as a rule, be not over 16 feet long, with a down spout at each end discharging on the ground. Do not use any cisterns if avoidable.

You need not pay any attention to open cesspools. You may have as many cesspools as you choose. These will breed Culex, but you will never find *Aëdes calopus* coming out of a cesspool. The *Aëdes calopus* does not like that kind of water; she requires clear water to breed in.

Another thing: How about the cups provided by the leaves and branches of plants? This may be matter for important decision sometimes. There are a number of plants that hold a little water in the axils of their leaves. A cornstalk does; so does the caladium which adorns our lawns. In the Tropics you will find plants that will hold some ounces of water, or even half a pint, and these may furnish breeding places for our mosquitoes, because the water is clear. The banana and taro (colocasia) should be especially noted, as the axils of their leaves may hold considerable water. The leaves of certain palms also form cups that hold water; some, as the coconut palms, very little, while the Traveler's palm will hold, maybe, half a pint. The colocasia, however, was our main concern on the Isthmus, as they were all around the houses and held two ounces of water or more to the plant. In this water the larvæ of *Aëdes calopus* was sometimes found, usually, however, in small numbers.

As I told you, I think, a mosquito lays by preference where her progeny is most apt to come to maturity, but she will lay by obliga-tion wherever there is water when she can not find a better place, as she can not help laying any more than any other female animal can.

To determine, then, if the water in these colocasia plants was suitable to bring the larvæ to maturity we put screens of wire or of hoops and mosquito netting (just the arrangement one uses to keep flies from cake) over these plants where there were collections of larvæ, and from what I saw I do not think 5 per cent of them developed into mosquitoes.

Bananas were examined and were not considered dangerous. I do not recall whether no larvæ, or very few, were found in them. These plants are not plentiful about the city of Panama. I do not know that any palms were examined. I think we had only two Traveler's palms on the Isthmus, and we did not fear the others. On the Isthmus, then, we did not consider the breeding in the axils of leaves of plants of sufficient sanitary importance to take measures against it. I can conceive, however, that the Traveler's palm may be a decided danger; as, indeed, under different conditions, the other plants mentioned might be. I am only telling you what the conditions were on the Isthmus; they might well be different elsewhere. It is fair to say, too, that on the Isthmus the removal of "cover" for mosquitoes in the neighborhood of houses removed most of these plants any way.

What I want you to notice here is not that we considered the breeding in plants of little sanitary importance, but the method of determining that it was not. To find larvæ in a collection of water is not proof that this collection is breeding. See if they develop into imagines before you say that.

Aëdes do breed in knot holes in trees. At Corinto the knot holes and hollows in the firewood which was piled on the wharf was a source which had to be reckoned with.

In artificial containers there are three methods you may adopt to get rid of mosquito larvæ or eggs: Turn the water out; screen the container; or, if these two are impracticable or not permissible, oil the container. This seems a simple thing to tell you, but it is a point you may have occasion sometime to remember. In turning the water out of the container turn it completely out; do not leave any water in it, or you will have to do your work over again. This mosquito has probably been turned out of water jars for generations. Those that have been turned out have not bred any progeny, and only the progeny of those that knew how to take care of themselves are alive. Thus the instinct to save themselves is strong in them now, and I must say that a great many of them seem to know how to take care of themselves. For example, of 100 larvæ in a jar that has been turned out, if you leave 50 drops of water in the jar you will find 99 of the original larvæ in the bottom; and the only way to make a good job of it is to turn it completely upside down and get rid of every drop of water that is in it. They cling to the last few drops of water left in the jar.

As to oiling containers, that, of course, can be done only when the water is not to be used for drinking or cooking purposes and where you can not turn it out. You will find this to be the only method available in a great many places. Salt, if enough be added, will also prevent breeding. This was the means used to get rid of them in water about a church which could not be oiled.

There is one thing you will have to bear in mind: If you turn a water supply out you have got to furnish other water for it, and this is not always easy to do. Supplies of wholesome water are often scarce, and in places where yellow fever is epidemic waterworks are usually conspicuous by their absence. In such places a man often has to buy drinking water as he would buy milk, at so much per gallon. You must not turn water out unless you can furnish other water. So, as a preliminary step, unless you are in a very great hurry, you should arrange to supply him with fresh water. That being done, I think you have a right to turn the water out of cisterns and pots. Otherwise people "won't stand for it," to use a slang expression.

The next general method for the elimination of yellow fever is the control of the human host. I told you, I think, that I regarded this as of small value where yellow fever is endemic and the people do not care. If I had a town like Guayaquil, for instance, I would not isolate a single man; I would not fumigate a single room; I would not go to the trouble of putting my finger on an infected mosquito, except for the purpose, maybe, of protecting a little foreign colony that might be in the place. I do not think any attempt from the human end would have any effect upon the time, by one day, when you would eliminate the fever. I would make my attempt entirely from the end of the insect. But it is very different in a town where yellow fever is not endemic. If this place be small, or if only a part of it be infected, and if you can have the cooperation of the people, as a rule you should make this the main attack, because it is possible to eliminate the fever in this way; and if so, you can do it much more quickly than by war on the mosquito. Even if you can not eliminate it you can always control it. And in those countries where it is not endemic frost will come after a while, and if you can keep it under control, lessen the rapidity of its spread, you can lessen the number of cases that will occur before frost stops it to a small proportion of what would have occurred had not these measures been taken.

In such a place, of course, there is no reason why you should not work from both ends, as White did in New Orleans and Gorgas in

Habana and Panama. I think I said all this yesterday, but the matter is so important that I repeat it.

I spoke of the cooperation of the people as being necessary for this work, and it is. You must work with the people; you must have their assistance if you hope to accomplish anything. You can not do efficient work if your activities are opposed by the community. I have tried it both ways and know.

Of course this method consists in finding the sick men, isolating them from mosquitoes, and destroying such mosquitoes as have had a chance to feed upon them before they were isolated. It hinges upon finding the sick man. If you can locate the disease and screen out the mosquitoes it is easy to destroy the mosquitoes that may have found access before you got there; but finding the sick man is not easy. You may have no difficulty in finding the bad cases, the cases that are known to be yellow fever. You have to bend every energy, and no trouble is too great, no precaution too trivial, to find the slightly sick man. By taking great pains in your search and by enlisting the support of the people, and through the laws and ordinances, you will find a great many.

The first thing to do is to lay out in your own mind the great fact that every man sick in a place where there is yellow fever is, from a sanitary standpoint, sick of yellow fever until the contrary is proved. I do not mean that every sick person actually has yellow fever. If, for instance, a man is reported as sick and you find he has a broken leg, you will know that his trouble is not yellow fever; but if you do not know that his leg is broken you had better look into his case and satisfy yourself just what the trouble is. And until you do know that the sick man has a broken leg he should be regarded as a case of yellow fever.

Sometimes you will find cases in which you can not tell whether they have yellow fever or something else. At McHenry, a little place in Mississippi, an epidemic broke out. It was a small place, but it was in June, and it was a place from which yellow fever could spread—a spark which could kindle a conflagration—and unusual precautions were justified. The town was divided into sections and every soul in it was seen twice a day. If any man was found sick he was seen by a physician and, unless the case was plainly not yellow fever, he was isolated. If anyone became sick at night or between visits in the day he was instructed to call a physician at once, and generally he did. In this way we kept a very close watch on the people in town and saw almost every case of sickness as soon as it occurred. To encourage the reporting of sickness not only was the family of the sick man put to no expense by his sickness; it and he were given all the help we could possibly manage. It was made an advantage to be sick, so as to get the sickness reported. Quite frequently we were unable to say when we first saw a patient whether he had yellow fever or not, and we did isolate quite a number who did not have yellow fever, yet I think we isolated all who did have yellow fever. It was very close work and under good conditions for success—a small place, with fair cooperation of the people and a condition which required unusual effort. It was successful and the first suppression of an epidemic of yellow fever in the South in the summer of which I know.

In Panama we had laws compelling the reporting of cases of sickness and we had physicians whose duty it was to see everybody who was reported sick; and, finally, we made a tabulated list of every one in town who was susceptible to yellow fever—the "nonimmunes"—and had each one of them inspected daily. Anyone missing had to be accounted for by his lodging-house keeper, and anyone even suspected of being sick was seen by a physician. The detail was as well worked out and as well carried out as we could do it. The town, however, was fairly large and some part of the susceptible people were opposed to our efforts—not actively, but opposed all the same. We did not find all of the sick, or enough of them to get rid of the fever.

I tell you these things merely as illustrations. I can not tell you how to go about finding sick men. That is a thing that has to be left to yourself, as conditions vary. You must use your own judgment and all the ingenuity you have, and, as I said before, you must regard every sick person as having yellow fever until the contrary is proved. At McHenry we had free medicine and attendance. We furnished these and some delicacies to everyone who was sick, and when you do this you will be surprised to find out how many report sick. If you make it hard for them you will never find them. To be sick must be made a privilege. Having found them, they should be removed to a hospital and screened, or screened in their own houses. It is better to take them to hospital if they are willing to go, but it is better to screen them at home if the knowledge that they will be taken to hospital will prevent your finding other sick people. It is better to find 90 per cent of the sick people and screen them in their houses than to find 50 per cent and screen them in the hospital; and it may be a question between isolating them in their houses or not at all. The killing of mosquitoes is a very easy thing; the finding of all the sick men is a very hard problem.

That, I think, is all that I am going to say about the sanitation of yellow fever. The principles I have tried to give you and some of the details. The others you will work out for yourselves according to the nature of your problem. And remember that each officer of this service always improves on his predecessor.

The clinical aspects of yellow fever I do not purpose going into, ıt the diagnosis of the fever, especially its differential diagnosis, a necessary point in the sanitarian's knowledge.

In the matter of diagnosis it is not worth while to say anything out the bad cases, the cases that anybody can tell. The cases that ill give you trouble are the light cases and cases in the early stages. arly diagnosis is especially desirable. Therefore it is the clinical story of the light cases and of those in their early stages that I am ing to try to give you.

In the first place, some cases are not diagnosable; you can not agnosticate them. Here is a family—the mother, the father, two der girls, 16 and 13, possibly a boy of 12, and some children of 5 6 and younger—all sick. The adults plainly have yellow fever, d some of the children's cases can be diagnosticated as such, but me of the little children are simply sick of a light fever for a few ys with no distinctive marks. It is reasonable to believe that all ve the same disease, but some of them taken by themselves could t have been diagnosticated.

Yellow fever commences just like any other fever. You have pains d malaise; may have a chill and again you may not. About half attacks commence at night. There is a feeling among southern ysicians that nearly all attacks of yellow fever commence at night: hink not. A little more than half the attacks commence at night, d as few malarial chills come on at night, a chill that comes at ht does help to exclude malaria. The pains and malaise are ater than you would expect from the elevation of temperature. hen you find a man with a temperature of 102° and sick enough 104° it is suspicious of yellow fever. It is so with some other eases—dengue and plague, for example. Headache is almost in- iable. I have seen only one patient that said at the time he had headache. It is generally severe. Pains may be moderate and degree is not of much prognostic import. I think I had the worst ns I ever saw in a case in the beginning, and I did not have a y bad case. Nausea and vomiting are rather more common than the onset of other fevers. Constipation is almost invariable, but bowels are not hard to move. Insomnia, or very troubled sleep, he beginning is, in my experience, universal. The patient is rest- and uncomfortable, more so than his temperature would lead you xpect. There is no albuminuria in the beginning. A slight leu- ytosis; otherwise there is nothing to note in the blood picture.

Iis facies in the very beginning is extremely characteristic, I do say "differential." It is not worth while to remember the "star- eyes" and all that is put in the books about it. Just remember t the first effect of yellow fever is to make an intense active capillary congestion of the skin and mucous membranes. The eye will, of course, be bloodshot; on account of the active congestion it will be watery. The upper lip will be puffed and swollen. The whole face will be slightly puffed and red; the gums red; the tongue red; fauces red. It is simply the effect of an acute active congestion.

One thing about the conjunctivæ: Guiteras, who is a most acute diagnostician, says that the conjunctivæ on the first day show yellow. But about 95 per cent of the men I have seen in the South have yellow conjunctivæ without yellow fever. An examination of all the white male patients in Ancon Hospital once gave practically 100 per cent with yellow conjunctivæ.

About the third day the "stage of stasis" comes on. This is the "stage of calm" of the old books, only the "calm" does not always occur. Dutrolau says it may be so short as scarcely to be observable, which I judge is about the same thing. Calm does not always occur, but *stasis* is invariable. The whole aspect changes. In light cases and those of moderate severity—it is only of those I am speaking—the pains disappear; the distress and restlessness are gone. The patient can sleep now or rest without sleeping. His appearance changes absolutely and is now not only characteristic, but distinctive. He lies flat in bed on his back; slides down on his pillow. The whole appearance of the man as he lies in bed is one of rest. His mind is clear; he speaks only when it is necessary to speak; speaks slowly, distinctly with a low voice; and he is tired, very, very, tired. I know of a case of moderate severity where the man had an electric bell lashed to his hand and when he wanted anything all he had to do was to press a button with his thumb, but he would wait until he almost perished of thirst before he was willing to undertake the muscular exertion of pressing the button. The nurse would come in and ask him what he wanted and he would say "water;" not another word. It was even too much exertion for him to say "drink," which was perceptibly more laborious to pronounce. That is how tired he was.

The appearance of the face changes. The active congestion of skin and mucous membranes of the first stage is replaced by a passive congestion—a stasis. The face is no longer swollen; it is not bright red; shows a dusky pallor rather; the conjunctivæ are red, but with tortuous veins; they are dry rather than moist, making the "dull-red eye" of this stage. A distinct yellow color is nearly always observable in the eyes. The gums are red, but dark red and spongy and are apt to bleed spontaneously or on slight pressure. The facies (the appearance of the patient) is now not only characteristic, but distinctive. At this stage the diagnosis can quite frequently be made from the facies alone. It can always be suspected.

If one presses the finger on the chest or draws the nail along the kin the white mark made will be obliterated very slowly, and after inally turning red will remain so for some time. The mark is not hite, by the way, but yellowish. This is the "tache" of the books, nd simply means a sluggish circulation and some jaundice. The tomach is frequently painful; always tender. It is very painful in evere cases and tender enough in cases of moderate severity, reuiring a cradle to keep off the pressure of the bedclothes. Having licited tenderness, be satisfied and do not press on the stomach any ore. This may produce vomiting.

Nausea, absent since the onset, may now return and is very apt to o so if food be taken. This, indeed, will likely lead to vomiting. omiting now if it occurs is a far graver symptom than that which ame on with the attack. It is in this stage that gastric and intes-inal hemorrhages occur, if they do occur.

Albuminuria is generally found on the evening of the third day. f it does not come on by the fourth day it is rarely found later.

Cases of moderate severity or a light case now proceed to get well, lowly at first. They are very weak at first. In a little while they ill gain, and when they begin to improve they will gain as rapidly s in any disease I ever saw, and will get completely well so far as know. They may turn markedly yellow in convalescence—eyes nd skin—or they may not, more often in convalescence than during e sickness. There is no constant relation between the gravity of e disease and this sympton, but grave cases are nearly always ellow. There is always, I think, some yellowness.

I have never seen any ill effects after yellow fever. Patients ther die from it at the time they have the disease or completely ecover. I never saw any sequellæ of yellow fever, although Carroll reported to have developed septic endocarditis from his yellow ever. I have known two physicians who believed they were peranently injured by it. Purnell died in 1909 of Bright's disease, hich he thought was contracted from yellow fever in 1879. I oubt its origin. It lasted a long time. Dr. Burr contracted the ever in the epidemic of 1868 at New Orleans, which he was sure ould shorten his life. He died in the spring of 1909, 73 years old. doubt that yellow fever had very much to do with his death.

In yellow fever there are three signs which merit fuller expo-tion: Albuminuria, pulse and temperature, and hemorrhage.

First, albuminuria. In the beginning the urine is like the urine of ny other fever, high colored and rather scanty, but on the second third day albumen appears. Of course, you have albumen in most any fever if you have fever enough, the agent causing the mperature reaction causing the albuminuria. But there are two points in the albuminuria of yellow fever which are of note. In the first place, it comes with very little fever and it *is out of all proportion to the fever.* When you find a man with a temperature of 100° or 100.5°, with a fair amount of albuminuria and fever lasting not over three days, you suspect yellow fever. When you find a man with a temperature of 101.5° to 102°, with albumen that you have to shake out of the tube, that is not the ordinary albumen of high temperature of an ordinary infection.

The second and more important point is that the albumen *increases with the fall of fever.* In the cases I am talking of the fever reaches its maximum in 48 hours. It will possibly run three days and commence to fall, and the albumen in these cases shows usually on the third day and increases on the fourth and fifth days. I found a case on the fifth day with a temperature of 101°, and the albumen had to be shaken out of the test tube. That class of albumen is characteristic and differential. That is the albuminuria of yellow fever.

The early appearance of albumen is a bad prognostic. When it appears on the first day it goes to a fatal termination; on the second day it is of very bad augury. Also, let me caution you to examine the urine when you first see your patient. Albumen is not caused by yellow fever in the beginning, but your patient may have albuminuria from other causes, and it is important to know whether he has or not. To find albuminuria on the third day and to have a doubt raised if it were not there before the present sickness is annoying; while to find it absent at first and then present in considerable quantities as the fever declines is of diagnostic importance.

Since, in spite of the severity of the nephropathy, permanent lesions of the kidneys are rarely, if ever, left, it is evident that the toxin of this disease must especially attack the tubular epithelium. I have never seen blood macroscopically in the urine of yellow fever.

Pulse and temperature.—Yellow fever is not a disease of high temperatures. Rarely does it go over 103.5° even in bad cases. The temperature of light cases is highest on the first day. The temperature of moderate cases is highest on the first or second day, and then commences to fall; 100° to 102.5° would include, I suppose, nine-tenths of all cases of yellow fever we get in this country. The temperature is a continuous one. You can not, by the temperature, tell when the stage of stasis comes on. From the symptoms the advent of this stage marks a crisis in the disease, but there is no crisis in the temperature chart. The temperature goes on gradually down to the fourth or fifth day or a little beyond the sixth day. Bad cases have it somewhat longer, and some cases go on for three or four weeks; but that is not yellow fever; it is some complication. We try to explain it, calling it "fever of secondary infection," which is as good a name as any other.

The pulse in the beginning is higher in proportion than it should be for the temperature. If you draw the pulse and temperature curves the pulse at first will probably run 10 to 20 beats more than it should for the temperature indicated. It then commences to drop. In most cases from the very beginning. The part of the abscissa included between the pulse and temperature curves increases as you go on. That is "Faget's sign." It is not always present, varying a good deal in different epidemics. It is, I think, less common in severe fevers. It was conspicuous by its absence—or rather rarity—in Panama. In Cuba it probably occurred in half the cases; in New Orleans it was much more common. Though not differential, it is a thing to look for. But you are quite apt to find the pulse falling away from the temperature in yellow fever. The lower the pulse within reason the better. I remember one man who had been quite sick and in convalescence I counted his pulse and found it 91 in three minutes, just about 30 to the minute, and he was doing very well.

Hemorrhagic signs usually appear first at the gums, for men at any rate. Examine the gums as soon as you see your patient. If the fever has just begun the gums, although bright red, will be of normal consistency. On the third or fourth day and later they should be soft, spongy, and bleeding on light pressure, or even spontaneously. Unless you examine them the first day you will not know the normal condition of the gums and could not then judge of the change that takes place in them, and it is this change you wish to know. Do not press the gums with the finger, or even with a handkerchief on the finger. This may cause gums which bleed easily—some people's do—to bleed independently of yellow fever. Press your finger lightly over the upper lip. Rarely will enough pressure be thus exerted to make the gums of a person in health bleed. Wipe it off the gums with a clean handkerchief. I am sorry to say that frequently you can not take the statement of your patient, or even of her physician, on the previous state of her gums if the fact that they are now bleeding is a point in the diagnosis of yellow fever. Hence, examine the gums as soon as you see her. Women and even female children well under puberty bleed from the genitalia. This for those over puberty is, I think, invariable. Capillary hemorrhages into the stomach and duodenum may occur during the stage of stasis and be shown either by the "black-vomit," beginning as small black or brown specks in the vomit, or as a tarry stool. The cases in which hemorrhages are a marked feature I need not describe; the laity will make your diagnosis.

The differential diagnosis should be plain to you from what I have said. What cases are you apt to mistake it for? Well, I have seen typhoid fever mistaken for it, but that was not necessary. I ha seen it mistaken for sunstroke. But you may, I think, legitimat mistake it for malarial fever, dengue, influenza, and measles. Y will laugh at the last one as one of the things it is apt to be mistak for.

Malarial fever.—The differential for malaria is, as a rule, fairly easy. Only do not lay too much stress on the plasmodia. If you d not find any plasmodia and the patient has not taken quinine, negatively the sign is of great value. Finding the plasmodium does no exclude yellow fever. Those of you who have been South will remember the outbreak of 1897 in Ocean Springs, where, because plasmodia were found in yellow fever cases, the diagnosis made was erroneous. We found malarial parasites in a large per cent of yellow fever cases from Cabanas, near Habana. They were not rare on the Isthmus. So the positive value of plasmodium is not of so much importance.

The time of onset is of some value. If the chill comes on in the night it is pretty certain not to be malaria, but if it comes in the daytime it does not exclude yellow fever.

The facies is distinct from malaria. A man would be unwise to make a diagnosis on the facies in the first stage, although he ought to be able to say that it was not malarial fever, and if malarial fever were the only alternative he would certainly be suspicious of yellow fever and count it yellow fever from a sanitary aspect.

The first year I was in Habana Maj. Gorgas and I used to go over to Cabanas early every morning to look over the sick people in the barracks there, and we used to write out the diagnoses of the men as we saw them. We saw, then, in the morning those who sickened in the night, within the first 12 hours of illness, so practically our guide was the facies. We were mistaken quite often, but rarely between yellow fever and malarial fever. Malarial fever plus alcoholism was less easy to differentiate from yellow fever.

Dengue.—Fortunately we can now tell dengue from yellow fever, and in the very beginning. It used to be very difficult, sometimes impossible, to tell one from the other in the early stages, and we had to wait. But the blood picture is absolutely characteristic, absolutely differential. Yellow fever gives a slight leucocytosis; dengue a profound leucopenia. It has been known to show as low as 2,500 leucocytes. I think in no other way can we be at all sure of differentiating between the two diseases in isolated cases in the beginning. Many people have written on the difficulty of telling the difference between the facies of yellow fever and the facies of dengue in the first stage of each, and each one has given his own minute and different points of distinction. There is *no* difference. The immediate underlying cause of the peculiar facies of the first stage—active capillary congestion—is the same for both, and naturally they both

esent the same appearance. When it comes to the second stage
is different. The facies, then, is sufficient for the differential diag-
sis, but we want to make the diagnosis early, and the blood picture
the differential between dengue and yellow fever or between dengue
d any other acute disease I can think of.

Influenza.—When catarrhal symptoms are absent it is sometimes
t very easy to tell influenza from yellow fever. I do not know how
u can do this except to wait for the second stage. We had one
se in Ancon Hospital that we held up until albumen appeared and
und it was yellow fever. We had another case that we held up and
ited till the albumen did not appear and found it was influenza.

Measles.—In the beginning this disease is very like yellow fever.
ne of the most acute diagnosticians I know of picked out a number
men from a regiment at Siboney as yellow fever cases that broke
t with measles three days later. The facies misled him, and I have
doubt that anyone else not thinking of measles among grown men
ght have fallen into the same mistake. Knowing the possibility
this error, Koplik's spots should save us even if we did not wait
r developments. I mention measles simply that you may suspect
asles.

Let me give you one last point in diagnosis: If you do not know
hether it is yellow fever or not—wait. You will know to-morrow
next day. Grave and damaging errors have I seen made by pro-
uncing a diagnosis when one did not know. It is a great pity not
be able to diagnose it in the first stage. But you had better not do
until the fourth day than diagnose wrongly. It is sometimes a
rious thing to diagnose a case of dengue as yellow fever and still
rse to say that yellow fever is dengue.

I wish I had time to tell you a little of pathological appearances
d the diagnosis from autopsy, which is at times not only important
t doubtful. I have not. I thank you for your attention.

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THE SOMATIC MITOSIS OF STEGOMYIA FASCIATA.

By LUCY A. CARTER, S.N.D., B.Sc.

Reprinted from 'The Quarterly Journal of Microscopical Science,'
Vol. 63, *Part* 3, *December*, 1918.

The Somatic Mitosis of Stegomyia fasciata.

By

Lucy A. Carter, S.N.D., B.Sc.

With Plate 21.

INTRODUCTION AND MATERIAL.

In the summer of 1917 Dr. Ashworth sent some eggs of Stegomyia fasciata to Prof. Graham Kerr, of Glasgow University, for museum purposes. These eggs had been obtained from females of a strain brought from Freetown, West Africa, by Mr. A. Bacot, F.E.S., Entomologist to the Lister Institute, London, successfully bred by him in London since his return.

Some of the eggs were incubated in the Laboratory of the College of Notre Dame where Sister Monica Taylor (D.Sc.) found their development to be unusually rapid, the fulfilment of a condition necessary for the successful study of somatic mitosis, in order that a copious supply of dividing nuclei may be secured in a single specimen—thus rendering the seriation of stages possible.

While awaiting the discovery of a technique which will overcome the difficulties of sectioning later stages of the eggs of Culex pipiens, in order to furnish material for tracing out the gradual evolution of parasyndetic conditions in Culex, she had suggested that I should investigate the somatic mitosis in another mosquito.

The arrival of the rapidly developing Stegomyia fasciata furnished material admirably suited for the proposed study.

Prof. Graham Kerr gave up his surplus supply and Dr. Ashworth very courteously added to his original gift by sending fresh batches direct to the College. I take this opportunity of thanking the donors for their kindness and liberality, and also record my thanks to Sister Monica for her advice and helpful criticism throughout.

This short investigation has, therefore, been made with a view to determining whether the pairing of the Chromosomes in the somatic tissues exists, and, if so, of possibly linking up by means of another species, with a less pronounced condition of parasyndesis, the egg of Culex pipiens with the later stages of development.

Very much work remains to be done on the passage of the telophase nucleus into the resting nucleus, and since in C. pipiens the telophases of the later spermatogonial divisions were observed to pass directly into the synizetic nuclei, it seemed probable, as already mentioned, that mosquito material would be favourable for such a study, as it has so proved.

Abundant cases of dividing nuclei were found in the body-wall cells, particularly in larvæ just about to undergo ecdysis. Tracheal tube cells, as well as cells of other tissues also showed occasionally many cases.

The methods used were the same as those employed for C. pipiens, i.e. small portions of the mosquito were fixed in the fluids known as Flemming, Gilson-Petrunkewitsch, in warm solution of corrosive sublimate in alcohol. (The fixative used for any particular study is indicated in the explanation of plates.) Sagittal sections 8μ thick were used exclusively, and all nuclei described and figured were wholly within one section. This precaution is necessary where the chromosomes are long as in this case.

The Flemming fixation has the effect of making the nuclei look larger. It does not, however, so clearly preserve the spindle apparatus, which appears to greater advantage in other fixatives where, at the same time, the chromosomes also stand out just as sharply defined and are as readily counted as in the Flemming. The stain was Heidenhain's hæmatoxylin throughout.

The gonad having the same topographical relations as in Culex, the third segment from the hind end of the abdomen was cut out and sectioned in order that the spermatogonial and oogonial mitosis might be investigated.

Both the ovary and testis are similar in structure to those of Culex pipiens, and need not be described in detail. The testis is divided up into a number of compartments, all the cells of one compartment being approximately at the same stage of division. As might be expected in the more rapidly developing species, a greater range of "spermatogenesis" stages is obtainable in one specimen—large early spermatogonia, smaller spermatogonia of the multiplication zone, primary and secondary spermatocytes, and complete spermatozoa often being visible in a single section.

The later stages of spermatogenesis follow quite closely those described for C. pipiens (**3**) (Pl. 21, fig. 29), and throw no special light on parasyndesis, hence they have not been included in this account.

Most probably because of its rapid development—the imago in some cases emerging from the pupal case seven days after the larva hatched—the synizetic nucleus which occurs so abundantly in the C. pipiens testis is not nearly so plentiful in Stegomyia, although, as will be shown later, the synizetic stages are analogous in the two mosquitoes. Thus the assumption that the synizetic nucleus of the male gonad functions as a sort of inactive phase in C. pipiens seems justified. Later it will be shown that the so-called synizetic nucleus in Stegomyia is really the stage characteristic of the nuclei of the two newly-formed daughter-cells, i.e. the stage following immediately on the telophase.

A synizetic-like stage in somatic—apart from spermatogonial and oogonial—mitosis was noticed in C. pipiens, but its connection with the telophase of a dividing nucleus could not be demonstrated. The ovary in the larva and pupa of Stegomyia is quite immature. It apparently grows rapidly and attains its full development when the imago has been suitably fed. Synizetic-like nuclei are much more numerous in the tissues of the immature ovary than in those of the testis. In the former the growth is relatively slower, and may be suspended altogether until the suitable diet has been supplied to the imago. Hence here the synizetic-like nucleus seems to function as a "waiting" nucleus.

There is no difficulty in determining four as the diploid number of chromosomes.

SOMATIC MITOSIS. (Passage of the telophase nucleus into the resting nucleus.)

I. SYNIZESIS. (Pl. 21, figs. 1, 2, 3*a*.)

As already indicated, the two masses of chromatin formed from four daughter-chromosomes which cap the pole of the spindle in late telophase become surrounded by a clear liquid as the nuclear membrane is completed round the daughter-products.

(N.B.—The nuclear membrane persists during mitosis as in C. pipiens.)

The spindle-fibres either disappear altogether or are drawn into the chromatin masses and the synizetic nucleus is to be seen in each of the two daughter-cells. These two nuclei become gradually separated one from the other by the growth of the cytoplasm surrounding them, but they can easily be detected until they have almost reached the resting condition. This account holds good for all the fixatives employed.

II. SYNIZESIS (i.e. late Telophase) TO RESTING STAGE (Pl. 21, figs. 3–6).

The telophasic mass becomes gradually invaded by the surrounding nuclear sap, and assumes a vacuolated appearance.

Strands of pale staining substance are now seen to connect the chromosomic mass with the nuclear membrane, and there is a tendency for the chromatin to be drawn towards the membrane.

In favourable specimens at this stage a condition similar in form to that of an early prophase can be detected (Pl. 21, figs. 3*c* and 4)—i. e. the chromatin has the appearance of two pairs of chromosomes—the members of the pairs being very much twisted one around the other. A comparison of Pl. 21, fig. 17 (an early prophase in a young testis where the number of compartments making up the whole gonad is twelve) with Pl. 21, figs. 3*c* and 4 brings out the likeness between a very late telophase and an early prophase, these interesting figures (3*c* and 4) illustrating undoubted cases of the telophase. There is, however, a difference in the staining capacity of the two stages, the early prophase showing a freshness and limpidity which is absent in the later stage of telophase.

Pegs of chromatin now begin to extend from the chromosomes, to lengthen and to anastomose, and as the chromosomes become more vacuolated the nucleus presents the appearance of a crown (Pl. 21, fig. 5), the nucleolus being conspicuous. Eventually the chromatin is fairly well distributed in blotches and patches, and a typical resting nucleus is the result (Pl. 21, fig. 6).

From the above description it would seem that there is a marked tendency for the chromosomes to emerge from the telophasic mass in two pairs, the outline of these two pairs being gradually obscured as the vacuolisation of the chromatin proceeds.

III. Resting Stage to Prophase.

At the outset of mitosis the chromatin assumes the character of a densely matted mass of very fine threads—difficult to stain—surrounding a characteristic deeply stained nucleolus (Pl. 21, fig. 7). In fact, this latter structure often looks like an irregular blob of chromatin; and since it gradually disappears as the new chromosomes become recognisable, it would seem as though it had acted as a storehouse of chromatin ready to furnish the prophase chromosomes.

The incipient chromosomes now begin to thicken up in places and to contract. Liquid collects in the nucleus, which becomes more distended in consequence, and this liquid apparently pushes out the newly-forming chromosomes towards the nuclear membrane (Pl. 21, fig. 8). In this, the early prophase condition, two very long chromosomes can be distinguished (Pl. 21, fig. 9). They are double, the two membranes of the pair being twisted about each other (Pl. 21, fig. 9). Gradually a condensation proceeds, the pairs untwist, but the degree of untwisting varies greatly. When the untwisting has been slight—i. e. when the full condensation of the chromosomes has been effected before the component chromosomes of the pair have succeeded in extricating themselves from their close approximation—then, in full prophase, there are apparently only two chromosomes (Pl. 21, figs. 11, 12, 13.) When the untwisting process is completed simultaneously with the full condensation of the chromosomes, then the members of the pairs lie side by side (Pl. 21, fig. 14). Sometimes the untwisting is completed before full condensation, when four chromosomes showing only slight parasyndesis are the result (Pl. 21, figs. 15, 16).

That faulty fixation has nothing to do with the varying appearance of two or four chromosomes is evident from the fact that the same phenomena occur whatever the fixatives employed, and in all the tissues—parasyndesis not being confined to any one or other of the somatic tissues.

There is no method of distinguishing the spermatogonial prophase from that of the primary spermatocyte. The degree of parasyndesis is always great in the gonad cells—the fully condensed chromosome always giving the impression of the haploid number. Studies made from the youngest compartment in young gonads composed of few compartments show a meiotic-like character of chromosome like that occurring in meiosis ring, S-shaped and 8-shaped figures being common (Pl. 21, figs. 17, 18).

These appearances in prophase are, however, followed by an anaphase which is typically somatic, and the nuclei can, therefore, be distinguished as spermatogonial in anaphase (Pl. 21, fig. 28). As in C. pipiens, the primary spermatocyte cells are spindle-shaped, whole chromosomes being separated in the first meiotic division (Pl. 21, fig. 29).

IV. METAPHASE AND ANAPHASE.

The two pairs of long chromosomes arrange themselves towards the centre of the nucleus, parallel to the plane of the future equator of the spindle (Pl. 21, fig. 19).

In the large cells, where the chromosomes are very long, i.e. longer than the equator, they accommodate themselves by twisting up in a zig-zag fashion. As a consequence of this they are often difficult to count. In favourable specimens of small cells, however, they can be distinguished, and the longditudinal splitting can be observed. Most probably because of this great length the usual V-shaped chromosome of anaphase is often replaced in Stegomyia by a Ѧ-shape (Pl. 21, fig. 25).

That there is a tendency for the homologous chromosomes to become clearly associated in their passage to the poles can be demonstrated in many cases of uncut nuclei where this association can in no wise be attributed to bad fixation. As a result, only two chromosomes can in such cases be detected at the poles (Pl. 21, figs. 23, 24, 27).

In other cases, however, the four chromosomes at the poles can be counted quite readily (Pl. 21, fig. 26), and that the members of homologous pairs are sometimes separated is evident from a study of the sizes of the chromosomes (Pl. 21, fig. 25).

As explained above, the long chromosome has a Ѧ-shape in anaphase; the short chromosome either a V or rod-shape. This alternate arrangement of Ѧ, with a rod at each pole of the spindle, must betoken a separation of the members of the pairs. In cases such as Pl. 21, fig. 25, the repairing of of the breaking asunder of the daughter-segments to their complete separation and arrangement in a ring (round the spindle axis) I would term the Metaphases."

This definition still holds good. It will be noted that Strasburger employs these terms Anaphase, etc., in the plural —Anaphasen, Metaphasen, etc.—thus indicating a series of changes in each case.

Parasyndesis (para, beside). The longitudinal pairing of the chromosomes—as opposed to Metasyndesis—introduced by Häcker, 1909, in "Die Chromosomen als Angenommene Vererbungsträger" ('Ergeb. u. Fortsch. der Zool.,' vol. i, p. 74). Thus: "For the pairing of the chromosomes I would, as already mentioned, keep the term Syndesis. When, in order to effect this union, the chromosomes arrange themselves (in pairs) with their long axes parallel, I propose to call this arrangement a Parasyndese (Paradese), or paradetic union. When the arrangement of the chromosomes is "end to end," the term metasyndese (metadese) or metadetic coupling would be more appropriate.

Prophase (pro, first). Introduced by Strasburger with Anaphase, q. v., thus: "The introductory phases of the nuclear division which I shall call, collectively, Prophases, begin with the appearance of the filament-skein (spireme). . . . Finally, the nuclear plate is ready to form, and herewith the prophases of the division terminate."

Resting Nucleus.—A nucleus in a state of vegetative activity, in which the chromatin is not arranged in the form of compact chromosomes; a nucleus "resting" from actual mitosis—the period between two successive nuclear divisions.

Synizesis (G. syn, with; hizo, place), introduced by McClung, 1905, in "The Chromosome Complex of Orthopteran Spermatocytes" ('Biol. Bull.,' ix, 1905).

It indicates the clumping together of the chromatin often observed in the meiotic prophase. It was included in Moore's earlier term, "synapsis."

Telophases (telos, end), first used by Heidenhain, 1894 in the "Neue Untersuchungen über die Centralkörper und ihre Beziehungen zum Kern und Zellenprotoplasma" ('A. m. A.,' xliii).

The closing phases of mitosis, from the massing together of the chromosomes at the two poles of the nuclear spindle up to the final establishment of the daughter-nuclei—i. e. to the end of the mitosis.

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EXPLANATION OF PLATE 21,

Illustrating Miss Lucy A. Carter's paper on "The Somatic Mitosis of Stegomyia fasciata."

[All figures were drawn with the Abbe camera under Leitz' $\frac{1}{12}$ in. oil immersion objective and Zeiss' compensating ocular 12. Magnification of 3500 diameters. The magnification of the figures as reproduced is shown by scale. Fixatives indicated: Gilson Petrunkewitsch, G.P. Flemming, F. Corrosive sublimate in alcohol (warm), C.A. All sections stained with Heidenhain's hæmatoxylin.]

Fig. 1.—Cell from gonad. Telophase passing into resting stage. G.P.

Fig. 2.—Nucleus passing into synizesis. Cell from body-wall. G.P.

Fig. 3.—Telophase—three stages *a*, *b*, *c*, from group of nerve cells. In *c*, two pairs of chromosomes coming out of the mass. G.P.

Fig. 4.—Interesting stage of somatic telophase, showing paired character of threads coming out of the telophasic-synizetic mass. Cell from the body-wall of fairly old larva. F.

Fig. 5.—Somatic. Synizetic mass unfolding; gradual conversion into resting nucleus; paired character of threads. F.

Fig. 6.—Two resting nuclei from body-wall. F.

Fig. 7.—Somatic. Very early prophase showing chromatin passing to chromosomes from nucleolus; from larva. C.A.

Fig. 8.—Early prophase from Spermatocyte I. Imago ♂. C.A.

Fig. 9.—Gonad cell showing double character of chromosomes. G.P.

Fig. 10.—Early prophase. Somatic cell from tracheal tube. G.P.

Fig. 11.—Prophase; two chromosomes—from ovary. G.P.

Fig. 12.—Somatic from body-wall. ♀. F.

Fig. 13.—Early full prophase in somatic cell from body-wall; chromosomes long and lying in the nuclear membrane. F.

Fig. 14.—Prophase; four chromosomes in two groups; cell from body-wall of fairly old larva. F.

Fig. 15.—As fig. 14.

Fig. 16.—Late full prophase; somatic; four chromosomes. F.

Fig. 17.—Cell from youngest compartment in a gonad consisting of twelve compartments; probably spermatogonial. F.

Fig. 18.—As in fig. 17. ♂.

Fig. 19.—Very early metaphase. Two long and two short chromosomes nearing the future equator. F.

Fig. 20.—Metaphase; somatic ♂; gonad has twenty-five compartments. F.

Fig. 20*a*.—Somatic from larva. C.A.

Fig. 21.—Metaphase; spindle threads well differentiated; apparently four chromosomes; from gonad ♀. G.P.

Fig. 22.—Early metaphase. Four chromosomes splitting longitudinally from body-wall of pupa. G.P. Faintly stained.

Fig. 23.—Metaphase showing chromosomes in groups of two; somatic cell from body-wall. ♀. F.

Fig. 24.—From gonad ♀, looks like two chromosomes. G.P.

Fig. 25.—Telophase. Somatic from fairly old larva. F. Two long daughter-chromosomes separated by the two short chromosomes.

Fig. 26.—Telophase. Somatic from body-wall of larva. C.A.

Fig. 27.—Telophase. Two somatic cells from alimentary canal wall of larva. C.A. The two long chromosomes have reached the poles, the two short ones are still at the equator.

Fig. 28.—Spermatogonial telophase from gonad (fifteen to eighteen compartments) of larva. G.P.

Fig. 29.—First meiotic division from pupa. F.

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TECHNIC FOR USE WITH HOMOPTEROUS VECTORS OF PLANT DISEASE, WITH SPECIAL REFERENCE TO THE SUGAR-BEET LEAFHOPPER, EUTETTIX TENELLUS (BAKER)

BY

WALTER CARTER
(Contribution from Bureau of Entomology)

Reprinted from JOURNAL OF AGRICULTURAL RESEARCH
Vol. 34, No. 5 : : : : Washington, D. C., March 1, 1927

PUBLISHED BY AUTHORITY OF THE SECRETARY OF AGRICULTURE, WITH
THE COOPERATION OF THE ASSOCIATION OF LAND-GRANT COLLEGES

U. S. GOVERNMENT PRINTING OFFICE : 1927

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Published on the first and fifteenth of each month. This volume will c
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TECHNIC FOR USE WITH HOMOPTEROUS VECTORS OF PLANT DISEASE, WITH SPECIAL REFERENCE TO THE SUGAR-BEET LEAF HOPPER, EUTETTIX TENELLUS (BAKER)[1]

By WALTER CARTER

ssociate Entomologist, Truck-Crop Insect Investigations, Bureau of Entomology United States Department of Agriculture

INTRODUCTION

So far as the writer is aware, no apparatus has yet been reported y means of which the virus causing curly-top of sugar beet can be eparated from both the insect vector and the host plant. In the ourse of studies of *Eutettix tenellus* (Baker) and its relation to curly-op, such an instrument was devised.

METHODS AND APPARATUS

At the beginning of these studies it was found necessary to evolve ome method by which the insect could be fed artificially. Pads of hick filter paper soaked with food solution were first used, the nsects being kept on them in Petri dishes. This method failed ecause the pads soon developed molds and could not be kept even noderately sterile. The activity of the insect also made difficult he frequent transfers that were necessary where the Petri-dish nethod was used. The next trials were with membranes made of ollodion on a cheesecloth base, these being sealed over the ends of test tubes containing the food solutions. In a few cases *E. tenellus* was induced to feed through these membranes for periods of several days. As a rule, however, collodion membranes become very hard and this method also was abandoned. Gelatin membranes were found to be too temporary in character and they provided a fertile substratum for fungous growth.

Through a local drug store an animal mesentery was obtained which is sold under the name of "fish-skin." It has been tested quite thoroughly and has thus far proved to be the most practical of any of the materials tried. No. 4 rubber stoppers are drilled out with as large a drill as can be used and a small circle of the membrane fastened over the hole in the large end of the stopper, either with shellac or with a glue made of automobile celluloid dissolved in acetone. Any waterproof adhesive will no doubt suffice. This stopper is then fitted into a short tube containing the food solution. A larger stopper holds the tube in place in a glass cylinder which serves as a cage for the insects. This arrangement is illustrated in Figure 1, A.

For the feeding of a large colony, or where mass action is desired, a small sack may be made of the membrane, which is then filled and hung free in a cage. (Fig. 1, B.) This modification is only of service where the concentration of the food material need not be

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[1] Received for publication Aug. 8, 1926; issued April, 1927.

Journal of Agricultural Research,
Washington, D. C.

Vol 34, No. 5
Mar. 1, 1927
Key No. K-167

30192—27

kept constant. With the smaller set-up (A) the evaporation
face is very small and little loss is experienced; furthermore,
equipment has the advantage of making more delicate tests poss

It has been found that the mortality with this technic is
high, but whether this is due to the inability of the insect to

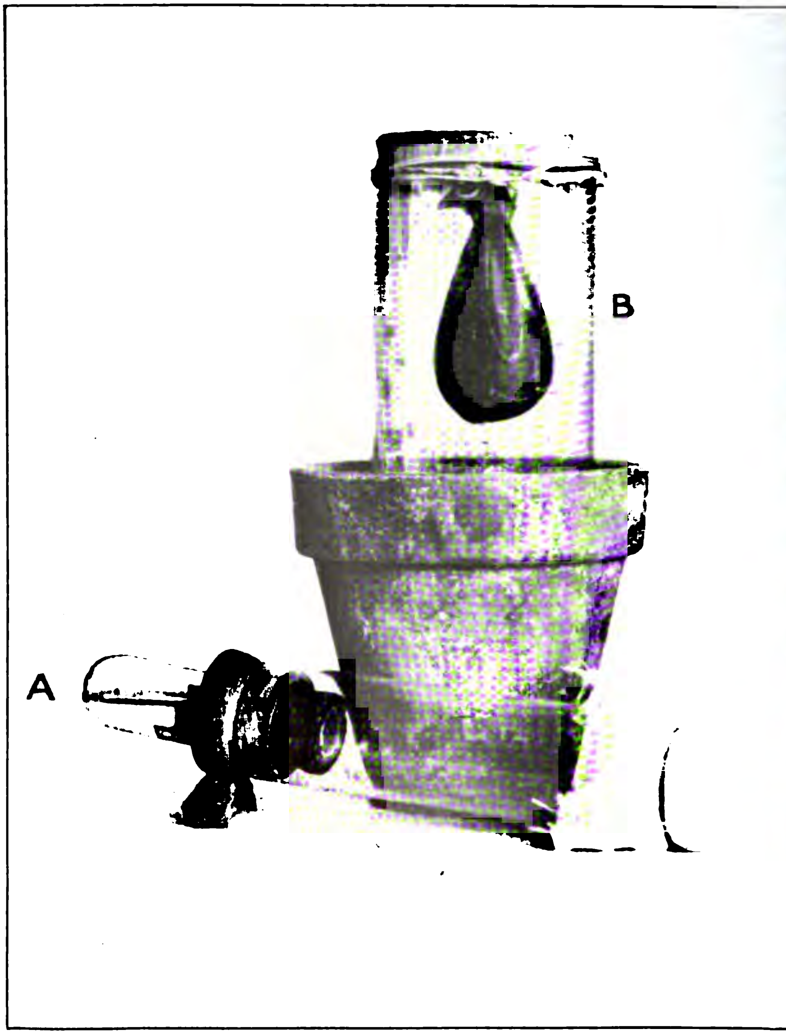

FIG. 1.—Apparatus for feeding *Eutettix tenellus*: A, set-up first used; B, modification of A, emplo
for feeding insects in large numbers (experience has shown this to be most practical)

the food or to the toxic nature of the food itself is difficult to de
mine. That *Eutettix tenellus* feeds through these membranes t
is no doubt since the feeding has been repeatedly observed, and
one occasion the membrane was peppered with tiny oozings f
the feeding punctures.

OLATION OF CURLY-TOP ORGANISM FROM INSECT AND HOST PLANT

A wide range of interesting possibilities is thus opened up, particu-ly, in this case, with regard to studies on the properties of the rus which the insect carries. For some time the writer did not ıd it possible by this method to produce the disease from the juice a diseased beet or from the sap of the wild host plants, but after any attempts the disease has been successfully transmitted from ice expressed from diseased beets to noninfective leaf hoppers and ence to healthy beets by means of this technic. The positive sult obtained by the writer was paralleled by similar results ob-ained by H. H. Severin, of the California Agricultural Experiment tation.

Extracted juice rapidly loses its original character and becomes oxic to the insect, so that it is not safe to feed *Eutettix tenellus* on uch juice for more than two or three days at 70° F. Tap water r weak sugar solutions have been the substances on which the writer as been able to sustain the life of the insect for the longest periods. *E. tenellus* fed for two weeks on a dilute cane-sugar solution and ne specimen of *Geocoris bullatus* Say was kept alive for a month on ap water.

Nutrition studies with sucking insects should be much simplified y this apparatus, since the quality of the food can be rather exactly ontrolled. Temperature studies are also facilitated, since the set-up an be used in connection with apparatus for maintaining controlled emperatures—a very difficult matter if it is necessary to feed the nsect directly on the plant.

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PROTEOCEPHALUS SANDGROUNDI, A NEW TETRAPHYLLIDEAN CESTODE FROM AN EAST INDIAN MONITOR LIZARD

WM. J. CARTER

Reprinted from Transactions of the American Microscopical Society
Vol. LXII, No. 3, July, 1943

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PROTEOCEPHALUS SANDGROUNDI, A NEW TETRAPHYLLIDEAN CESTODE FROM AN EAST INDIAN MONITOR LIZARD[*]

WM. J. CARTER

The material used in this study comprises one of several lots of tapeworms from the Dutch East Indies collected by Dr. J. H. Sandground formerly Curator of Helminthology, Museum of Comparative Zoology, Harvard College) and kindly presented to Professor Hughes. The label with the worms bears the following data: "*Varanus komodoensis* . . . Loc. Komodo . . . VII 1923 . . . Ex Mus. Buitenzorg . . . now in alcohol." Buitenzorg is in western Java; an island by the name of Komodo lies east of Java and south of Celebes.

For study the specimens, consisting of several fragments of strobilae without scoleces, were stained in borax carmine and mounted *in toto* in Clarite.

Proteocephalus sandgroundi n. sp.

(Figs. 1–6)

Description. Maximum width 2.15 mm. Largest fragment of strobila 175 mm. long with 158 proglottids all of which contain eggs; another fragment, 77 mm. long, has 96 segments none of which are gravid. It would seem that a complete strobila would be at least 300 mm. long. Segmentation distinct but nowhere pronounced. Margins, nearly smooth and straight; sometimes slightly bulged and sometimes slightly incurved or notched at boundaries between segments. Strobila very thin. Segments generally all somewhat wider than long although in stretched specimens the reverse may be true. No scolex, neck, or very young proglottids present in the material. Mature segments squarish, usually slightly wider than long, 1×1.2 mm. Gravid segments 0.94–1.57×2.07–2.15 mm. Ventral excretory canals comparatively broad and nearly straight; the dorsal ones narrow and very tortuous; no transverse connections observed.

Genital pores irregularly alternate; not at all salient; generally precisely marginal and usually within posterior third of body although occasionally almost as far forward as middle of body; about 45μ long. Genital atria very shallow. Cirrus pouch transversely elongate; somewhat constricted near the middle, the medial end being a little the larger; 233–246×53–66μ in mature proglottids or from one-fifth to one-fourth as long as width of segment. Cirrus when fully protruded about 55μ long; unarmed; slenderly conical it tapers toward truncate tip. Vas deferens somewhat coiled or looped within medial end of pouch; extensively convoluted beyond pouch,

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[*] Contribution No. 112 from the Department of Zoology, Oklahoma Agricultural and Mechanical College; prepared under the direction of R. Chester Hughes.

the general course being directly mediad; traceable as a single tube inward from pouch to median line. Testes 107–115 in number; spheroidal to ellipsoidal; 52–67μ in maximum diameter in mature segments; broadly distributed in a single layer between excretory canals on opposite sides an from region of ovary to front margin of segment; separated in two distinc lateral fields in mature and older segments; in approximately equal numbe in the two fields.

Vagina narrow, nearly uniform in diameter; only slightly undulant i follows a rather smooth course, running at first mediad, thence curving toward shell gland; its distal portion apparently always behind cirrus pouch. Ovary near posterior margin of segment; distinctly bilobate; 596–682μ in transverse diameter by 128–137μ in maximum longitudinal breadth. Lobes of ovary narrowly elongate transversely; pointed distally and broadest near their medial ends; with arched anterior, and straight posterio optical margins; entire and of smooth outline at maturity each lobe late becomes dissected into a grape-like cluster of tiny lobules. Shell glan clearly recognizable; situated posteriorly between lobes of ovary. Vitellari finely follicular, in narrow submarginal bands that extend entire length o segment; poral band dorsal to, and slightly reduced at level of, cirrus pouc Vitelline ducts arise at very near the posterior ends of vitelline field Uterus, at first a simple slender median canal spanning almost entir length of segment, later develops 10 to 20 pairs of somewhat overlappin lateral branches and occupies most of medial half of proglottid. Eg broadly ovoidal, 30–35μ in maximum diameter, with light amber-color chitinous shells; arranged in more or less scattered clumps or balls of fro four to over a dozen eggs each.

Host. "*Varanus komodoensis.*"

Locality. Komodo, Dutch East Indies, at about 119.3 degrees east lon tude and 8.5 degrees south latitude.

Cotype specimens. In toto mounts of several fragmentary strobilae, United States National Museum and in collection of Dr. J. H. Sandgrou

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EXPLANATION OF PLATE

The figures all represent *Proteocephalus sandgroundi* n. sp. and were all made with aid of a camera lucida.

FIG. 1. Mature segment, dorsal view. Dorsal excretory canals in single solid lines; side ventral canals in broken lines.

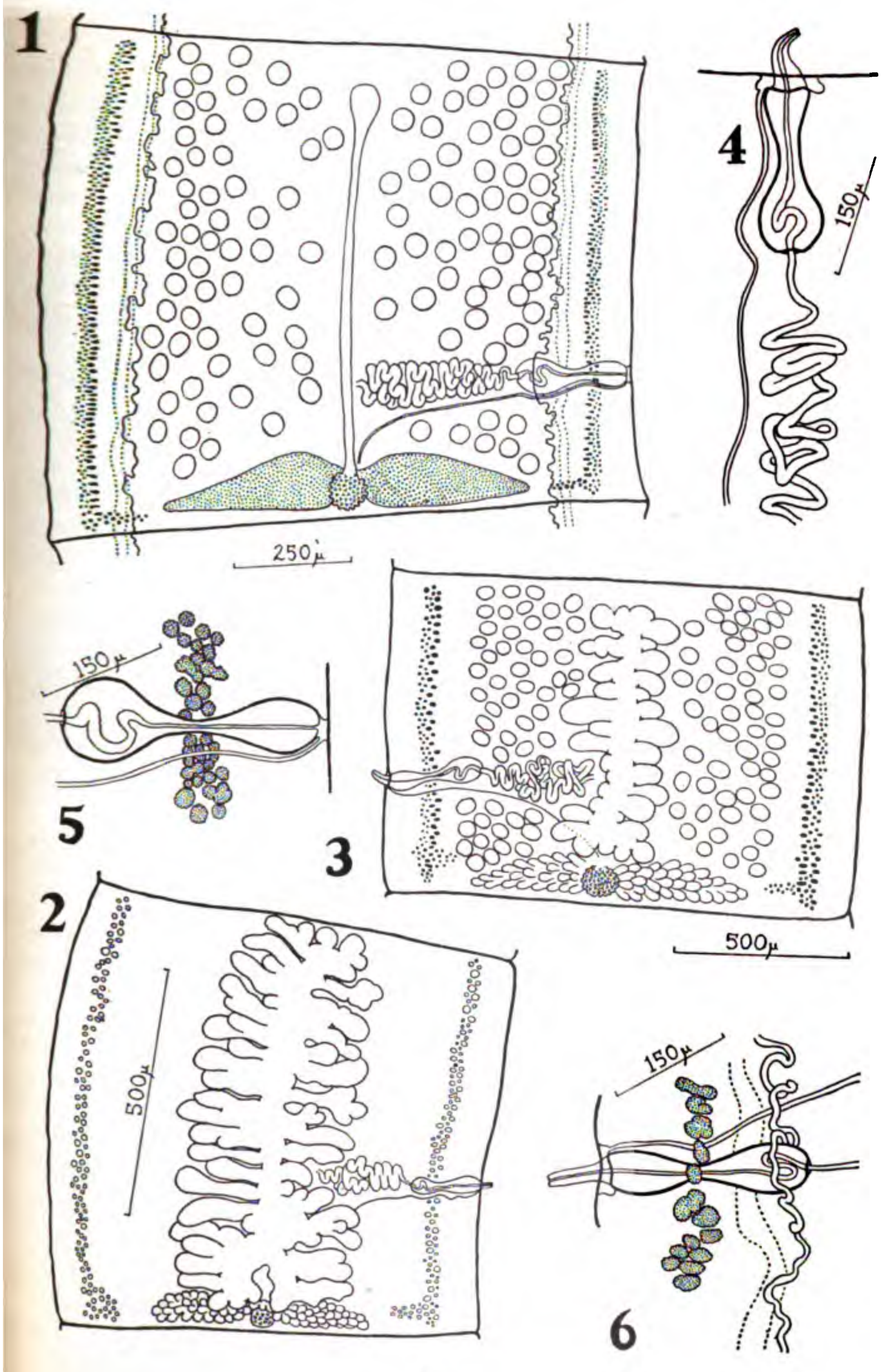
FIG. 2. Fully gravid segment. Testes, although still faintly visible, not included.

FIG. 3. Partially gravid segment.

FIG. 4. Distal portions of genital tracts. Cirrus everted. Anterior side to the right.

FIG. 5. The same, ventral view. Cirrus retracted.

FIG. 6. The same, dorsal view. Cirrus everted. Posterior side above.



PLATE

Discussion

The very large genus *Proteocephalus* Weinland 1858 comprises spec
that utilize cold-blooded gnathostomes as definitive hosts. Hughes, Bak
and Dawson (1941) listed 61 recognized reptilian species 13 of which occ
in lizards. The new form was studied in comparison with descriptive da
on and found to differ appreciably from all of the previously known re
tilian species, with the exception of two described by Hilmy (1936). R
peated efforts to obtain Hilmy's paper have been in vain. It will perha
suffice here to consider the relationships of the new form to the oth
saurian species—our sources of information on the latter being compilato
records in La Rue (1914), Rudin (1917), Baer (1927), and Meggitt (192
for the older ones and the original accounts of all described since 191
Differing from *Proteocephalus sandgroundi* n. sp. mainly with regard
features severally indicated the other lacertilian species are:

(1) *P. amphiboluri* (Nybelin 1917) Hughes, Baker, and Dawson 19
has considerably larger testes, an armed cirrus, a conspicuo
vaginal sphincter, and the distal portion of the vagina often locat
in front of the cirrus pouch;

(2) *P. articulatus* (Rudin 1917) Baer 1927 has the genital pore locat
anterior to the middle of the segment, the cirrus pouch relative
much longer, the cirrus armed, and the testes fewer in numb
and distributed in a single field;

(3) *P. beddardi* Woodland 1925 has more pronounced segmentatio
the cirrus armed, the female genital pore often anterior to the ma
pore, and fewer testes;

(4) *P. biròi* (von Rátz 1900) Johnston 1911 is much smaller and h
the genital pores farther forward, ripe segments twice as long
wide, fewer testes, and very vague segmentation;

(5) *P. continuus* (Rudin 1917) Baer 1927 has fewer testes, farthe
forward genital pores, armed cirri, and indistinct segmentatio

(6) *P. gracilis* (Beddard 1913) Hudson 1934 is considerably small
with fewer testes and ripe segments thrice as long as wide;

(7) *P. niloticus* (Beddard 1913) Baer 1927 is much smaller wi
farther-forward genital pores, longer-than-wide ripe segments, a
indistinct segmentation;

(8) *P. saccifer* (von Rátz 1900) Baer 1927 has fewer testes, grav
proglottids twice as long as wide, and a much smaller general siz

(9) *P. shipleyi* (von Linstow 1903) Baer 1927 is much smaller wi
genital pores farther forward and fewer testes;

(10) *P. striatus* (Johnston 1914) Hughes, Baker, and Dawson 1941 h
fewer testes, the vaginal pore anteroventral to the cirrus pouc
and the genital pores farther forward;

(11) *P. tidswelli* (Johnston 1909) Johnston 1911 has a much more slender strobila, the older segments much longer than wide, and fewer testes;

(12) *P. varius* (Beddard 1913) Baer 1927 is a much smaller worm with fewer testes and ripe segments four times as long as wide; and

(13) *P. woodlandi* Moghe 1926 has a much smaller strobila, a more prominent genital atrium, the distal portion of the vagina often dilated and often anterior to the male tract, a conical cirrus pouch, the uterus at maturity not extending so far forward, and the eggs scattered separately in the gravid uterus.

The above-enumerated species are all of the Old World. Nos. 2, 5, and 7 occur in Africa; 3, 4, 9, and 13 in India; 1, 6, 10, 11, and 12 in Australia; and 8 (*P. saccifer*) in New Guinea. All except *P. amphiboluri* and *P. striatus* were described from varanid hosts; apparently none of them, however, have been reported from *V. komodoensis*. Baer (1927) regards *P. articulatis*, and *P. continuus* probably, as being identical to *P. niloticus;* he also doubts that *P. woodlandi* is distinct from *P. shipleyi*.

Noteworthy features of the new species include the shallowness of the genital atria, the hourglass form of the cirrus pouch, the far posteriad positions of the genital apertures and vitelline ducts, the slenderly pointed form of the ovarian wings, the clumping of the eggs, and the chitinization of the egg covering.

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By

Lewis R. Cary (Princeton, New Jersey U. S. A.).

With 4 Plates.

Abdruck
aus den
Zoologischen Jahrbüchern.
Herausgegeben von Professor Dr. J. W. Spengel in Gießen.
Abteilung für Anatomie und Ontogenie der Tiere
Achtundzwanzigster Band. Heft 3.
Verlag von Gustav Fischer in Jena.
1909.

THE UNIV
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Übersetzungsrecht vorbehalten.

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With Plate 30—33.

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Introduction.

On examining specimens of the fresh water snail, *Goniobasis virginica*, brought into the laboratory in September 1908, it was found that practically all of them were heavily infected with the sporocysts or rediae of a trematode. This species of snail is very abundant in the canals and rivers throughout the region about Princeton. So, at the suggestion of Prof. Conklin, I undertook a study of the nature of the development of the germ cells in the nurse generations of this form, the wealth of material available offering an exceptional opportunity for the solution of this problem.

Since only the generations — Sporocyst and Rediae — from the secondary host were known, experiments were undertaken to determine the life history. When the adults were finally secured and their identity established, it was found that only this stage in the life cycle had been described, so it was thought best to describe and figure all of the stages; the more especially since the rediae and cercariae differ very markedly in many characteristics from the corresponding stages of the European form, *Amphistomum* (*Diplodiscus*) *subclavatum*, with which the species under consideration has been confused by all authors previous to Stafford (1905).

The work of which this paper is an account, has been carried on in the Zoological Laboratory of Princeton University during the session of 1908—1909.

I take pleasure in expressing my sincere thanks to Prof. E. G. Conklin for his unfailing interest, and for many helpful suggestion which have been of great value in carrying out this research.

Material and methods.

The greater mumber of infected snails collected were secure from two localities: first, the Deleware and Raritan canal, nea Princeton; and secondly from the Deleware River at Trento Practically all of those from the first mentioned body of water co tained rediae, in the most of which were found well develope cercariae. Those from the Deleware River, on the other hand, wit few exceptions, contained sporocysts, and inside these unsegmente eggs and young embryos (Germ Balls) were found in abundance. should be pointed out that in the, snails from the last mentio locality the germ balls within the sporocysts were giving rise cercariae and not to rediae. The greater number of infected sna taken from the canal during the spring months also contained sporocysts in which cercariae were developing.

All of the adult worms obtained during the early part of this study came from artificially infected tadpoles kept in the laboratory, or from frogs which had been fed with the intestines from such tadpoles; but in the spring many adult worms were obtained from frogs taken from ponds near the canal where infected snails were abundant.

The material both for sections and whole mounts was fixed with a number of reagents: FLEMMING's stronger and weaker solutions, HERMANN's fluid, BOVERI's picro acetic, picro sulfuric and sublimate acetic were used. HERMANN's fluid and Picro acetic gave the best results for material to be sectioned. For material to be used as whole mounts the picric acid mixtures were the most useful.

When intended for sectioning the worms were fixed while still within the liver of the snail, as it was found that there was less likelihood of distortion of the tissues when this method was followed, than when they were separated from the snail liver and preserved by themselves.

The material for whole mounts was shaken free from the snail tissue in a dish of water and killed in one of the picric acid mixtures. For staining this material CONKLIN's Picro-Haematoxylin gave the best results. The snail livers containing the parasites were imbedded in paraffin in the usual manner and serial sections 2, 3 and 5 micra in thickness were cut from them. The first mentioned thickness (2 μ) was the one used for all of the material which contained the earliest stages in the development of the germ cells.

After a few unsuccessful trials of other stains, HEIDENHAIN's ron Alum Haematoxylin, followed by a counter stain of Eosin in 5 $^0/_0$ alcohol, was used for all the sections, whatever had been the uid used for their fixation.

For the study of the youngest germ cells smear preparations made from a fragment of tissue from the broad end of the sporocyst were attempted, but none of these preparations gave as satisfactory esults as were obtained from the sections, so this method was bandoned.

For studying the living material of all stages aqueous methylin lue and neutral red were sometimes useful, although these stains ere of little use in differentiating tissues other than the nervous nd glandular structures.

Part I.

Descriptive.

A. The adult worm.

The adult worm (Figs. 1 and 2) has been recorded as occu in the cloaca of Frogs, Toads and Newts taken in many locali in North America. Leidy (1856), Pratt (1903) and Stafford (1 referred this form to *Amphistomum* (*Diplodiscus*) *subclavatum*. last mentioned author, in another publication (1905) establishe new species, *Diplodiscus temporatus*, for this form and gave a s account of the most striking features of its anatomy.

The sexually mature specimens vary in length, when fully tended, from 2 mm to 6 mm. The average length is about 3,5 In such a specimen the width of the posterior sucking disc (Ac bulum) is 1,4 mm; that of the body just anterior to the acetabu 1,2 mm. The worms move about so actively that accurate meas ments while they are alive are obtained with difficulty, while being killed, even though they are previously narcotized, the traction is so great that the measurements made from preser material are very misleading.

The anterior end of the worm is narrow for about one thir the entire lenght. From this point the width increases quite rap to the extreme posterior end, at the junction of the acetabulum

The anterior sucker is situated at the end of the body opens forward. The large posterior sucker opens backward downward, so that the worm is considerable longer on the do than on the ventral surface.

The body is covered with a thick, smooth, cuticular layer.

Muscular system.

The body wall has the usual systems of muscles; circu longitudinal and transverse. There are two sets of the last mentio fibres running in opposite directions. The circular muscle fibres extremely minute. Those of the longitudinal muscles, and of radial muscles are about twice the size of the circular fibres. none of my sections of adult worms, or cercariae, did I succeed demonstrating the muscular structures which have been descri and figured by Bettendorf (1907) as occuring in several forms the Malacocotylea, and in *Polystomum* among the Heterocotylea.

Digestive system.

The mouth, which is situated at the centre of the anterior sucking disc, opens backward into two pharyngeal pockets, one on either side. These are situated dorsally to the level of the mouth cavity. Posteriorly the mouth cavity opens into the long narrow oesophagus, which extends backward about one third the length of the body. At its posterior end there is a pharynx-like swelling with comparatively thick muscular walls. This expanded portion of the oesophagus opens into paired caeca. These extend along the sides of the body nearly to the posterior sucking disc.

The wall of the intestine is made up of a layer of large vacuolated cells which rest on a thin basement membrane. Each cell contains a large nucleus, which is situated in the basal portion of the cell, where the cytoplasm is most homogeneous. From the inner ends of the digestive cells there is a rather ill defined layer of fine protoplasmic processes which extend into the lumen of the gut. Ziegler (1883), Looss (1894), Tennent (1905) and other investigators have described the same condition in the intestinal epithelium of many trematodes. These structures are usually considered to be of some service in the absorption of food.

Water vascular system.

The main trunks of the water vascular system are clearly seen in the living worm. The terminal cells (flame cells) are readily demonstrated when a living worm is put under pressure beneath a cover glass and examined after it has stood for from thirty minutes to an hour.

The excretory pore is situated on the mid-dorsal line just anterior to the border of the posterior sucking disc. From this pore a short canal leads forward and downward to the excretory bladder, which lies in the dorsal part of the body between the ovary and the acetabulum. In a living worm the excretory bladder may be seen to contract rhythmically as long as the animal remains at all active.

The two main lateral trunks, one on either side, extend from the bladder, at first laterally, until they approach the body wall. Here each turns anteriorly and runs along beside the caecum of the corresponding side. In its posterior half each main trunk is much convoluted. The folding in my specimens was about equal in the dorso-ventral and lateral planes. Some of the folds lie above caecum, others below.

Stafford (1905) in the original description of this species d not mention any lateral foldings, he says: "From it" (The excret bladder) "below, spring right and left lateral ducts, which pass be the ends of the caeca and fold up and down along the outside the latter on their way forward to the region of the pharyng pockets. Two large lymph vessels lie along the inner, lower si of the coeca and posteriorly approach each side of the bladder remain distinct from it".

Anteriorly the lateral trunks extend to near the region of pharyngeal pockets where each one, after bending backward, bre up into a system of anastamosing vessels which extends to all pa of the body. At its end each one of these branches receive number of small „collecting tubes" at the distal end of each which is situated a flame cell.

Nervous system.

The nervous system in *Diplodiscus temporatus* conforms to type generally found in the Malacocotylea. The central nerv system consists of a pair of cerebral ganglia which are situated j posterior to the pharyngeal pockets, and which are connected b broad supra-oesophageal commissure. From each cerebral gangl a number of longitudinal trunks arise. One of these on each s runs anteriorly and supplies the organs in that part of the bo Posteriorly three large trunks are given off from each ganglion. these one pair is dorsal, the trunk from each ganglion being pla quite near to the median line. Another pair are lateral in positi These run along outside the water vascular trunks on each side the body. The third pair of nerve trunks lie in the ventral p of the body. As regards their distance from the median line, t are intermediate in position between the dorsal and lateral pairs

The three pairs of nerve trunks are connected by a comp system of commissures. Just posterior to the brain there is a sm commissure connecting the dorsal pair. Further backward th are a number of large commissures which completely encircle body. At the posterior end of the body the dorsal and vent nerve trunks of the two sides bend inward and each pair beco connected. Branches are given off from the lateral trunks, and fr the commissures, throughout their entire length.

Reproductive organs.

The testes are situated in the anterior half of the body; the anterior to the right, the posterior to the left of the median plane. In none of the specimens which I have examined is there a fusion of the testes to form a single mass, as has been described and figured by PAGENSTECHER (1858), WALTER (1858) and LOOSS (1892) for *Amphistomum subclavatum*. From each testis there comes a short vas deferens the two of which join to form the common seminal duct. This organ is considerably coiled. It passes anteriorly into the ductus which is provided with a prostate gland. The penis sheath is small and thin walled. The genital pore is situated on the ventral surface just beneath the point where the two caeca join together.

The ovary is situated in the median plane, a little way behind, and dorsal to, the posterior testis. It is considerably smaller than one of the testes. The shell gland lies just behind the ovary. A LAURER's canal runs upward and slightly forward to the dorsal surface of the body. The uterus, which in the young adults is ribbon-shaped, passes first backward from the ootype, then bends forward and makes several coils before reaching the genital pore. The vitellaria in this species are short in comparison to those of *Amphistomum subclavatum*, which extend forward nearly as far as the anterior ends of the main excretory trunks. In *D. temperatus* they extend from about the level of the posterior testis backward to the end of the caeca. There are from six to ten follicles in each vitellarium. The anterior follicles are above, the posterior ones below the level of the caecum. The vitelline duct comes off from the centre of each vitellarium at the point where it bends around the inside of the caecum in its passage from the upper to the lower side.

B. **Larval forms.**

1. The sporocyst.

It has been impossible to make any observations on the development of the eggs from the adult (bisexual) generation, or the infecting of the snails by the free swimming larvae.

The youngest sporocysts found in the snails were nearly spherical sac-like bodies about 0,1 mm in diameter. There was no apparent differentiation of any axes by which one part of the body could be recognized as anterior or posterior. In sections (Fi the body wall is seen to be made up of a very thin cuticular l immediately beneath which there are a number of muscle fi composing two systems. When a well defined axis becomes recog able these two sets of muscles are seen to be longitudinal circular respectively. Inside the muscular layers there is a l of cytoplasm containing a number of nuclei, but there are as no cell walls. The body cavity contains germ cells (eggs) in e stages of development.

In somewhat later stages the sporocyst acquires the chara ristic form shown in (Fig. 11). The broad end — posterior — alw contains the ovary (Keimlager) when this has become differentia In some instances the older sporocysts are surrounded by a mor less well defined layer of cells, the "Paletot" of early writers. material clearly shows that, as has been pointed out by Schwa (1886), this layer is made up of the leucocytes of the snail an not a part of the sporocyst. In some instances there is some evid that a division of the sporocyst is taking place; but not a si instance has been observed where this could be determined certainty.

Sections through the wall of older sporocysts show the o layers in practically the same relation as that described for earlier stages. In the structure of the inner layer, however, s very important changes have taken place. These will be descri in detail in a following section of this paper treating of the or of the parthenogenetic eggs.

2. The redia.

The youngest rediae found were about 0,5 mm long and 0,2 in diameter. These had already attained the characteristic sh of the redia, and the pharynx, gut, and body cavity were alre marked out, though not definitely established. The mature re (Fig. 4) are about 1 mm long and 0,3 mm in diameter. The ante end is broadly rounded, while the posterior end is sharply poin This part of the body is very active during life, often becom drawn out into a narrow process which is continually moving f side to side. At a point about one fifth the length of the body f the posterior end there is on each side of the body a lateral growth; the so called „posterior locomotor appendages“.

These last mentioned structures, which are always presen the redia of *Diplodiscus temporatus*, seem according to Looss' (1892) figures to be entirely wanting in the redia of *Amphistomum (Diplodiscus) subclavatum*. His figures (9 and 10 tab. 20) show the redia of that species to be comparatively narrow at the anterior end, from which it increases rapidly in diameter for about one fifth of its length. From this point it tapers quite regularly toward the posterior end which (fig. 10 tab. 20) is sharply pointed. The intestine is very short, and flask-shaped, in all of the developmental stages of the rediae figured. In *D. temporatus* the gut is comparatively long, frequently extending nearly the entire length of the body cavity. It usually lies close against the body wall along the ventral side of the redia.

ROSSBACH (1906) has called attention to the fact that the early stages in the development of the redia of *Amphistomum subclavatum*, as figured by Looss, differ very much from the conditions found in most of the other species of Distomae. The youngest redia figured by Looss, which is as yet (fig. 1 tab. 20) only a small sac-like structure scarcely one fourth the length of the fully developed redia, is shown with a large body cavity in which are contained several embryos. The largest of these is made up of about twelve cells. The "Keimlager" is shown to be already established as a thickening of the inner wall of the posterior end of the body.

The digestive system, on the other hand, seems, so far as I can make out from the figure, to consist of an undifferentiated mass of cells. There is no apparent distinction between the primordium of the gut and that of the pharynx, while no trace of the lumen of either of these organs can be made out from the figure.

In *Diplodiscus temperatus* the development of the redia takes place in the manner which has been described by ROSSBACH for the rediae of several species of Distomae. In the youngest rediae among my material there was a series of cells constituting the primordium of the gut which was becoming seperated from the surrounding cells (Meristem cells of SCHWARZE) by a well defined space, the beginning of the body cavity. At the anterior end of the redia the pharynx was established and had already become hollowed out. The body cavity was very small. The surrounding wall was thick on all sides, especially at the posterior end of the body. There were no embryos in the body cavity, nor indeed any cells (nuclei) in the body wall which could with any certainty be distinguished as germ cells at this stage in the development.

3. The cercaria.

The development of the cercaria will be discussed in a la section of this paper. The present description will, therefore, confined to the fully developed cercaria at the time of its lib tion from the sporocyst or redia.

The mature cercaria (Fig. 6) is about 2 mm, long — including tail -- and 0,15 mm in its greatest breadth. It is elongate ova shape and flattened in the dorso ventral direction. The tai quite slender and is about three fourths the length of the body

The cercariae are very active when first liberated from redia or sporocyst, and are constantly altering their shape. Swimm is accomplished by means of a vigerous lashing of the tail, sup mented by a rhythmical bending of the body. That this mentioned process is sufficient to bring about progress through water is established on the examination of almost any lot of li cercariae. It will be found that some of the actively swimm larvae will have lost their tails, but that in spite of this fact are able to make quite rapid progress through the water. anterior end of the body is always directed forward in swimm whether or not the tail is present.

The living cercariae are colorless when viewed by transmi light and are very transparent except over the areas occupie some of the denser organs.

At the anterior end of the body there is a small sucking The posterior sucking disc is situated on the ventral side o body a little posterior to the centre.

The body is covered by a thin cuticular layer.

Digestive system.

The mouth is situated at the anterior end of the body at centre of the anterior sucking disc. It opens backward into r and left pharyngeal pockets. From these a narrow oesophagus backward for about one third the length of the body. The o phagus ends in a pharynxlike swelling which in turn opens int paired intestinal caeca. These extend backward nearly to posterior end of the body.

Excretory system (Water vascular system).

In the living cercaria the main trunks of the water vascular system are the most prominent structures in the body. They appear as two large tubes, about three eights as large as the intestinal caeca, and are made up of large cells with highly refractive nuclei. The excretory bladder is situated near the posterior end of the body and is connected by a short canal with the excretory pore which lies just anterior to the tail on the dorsal surface of the body (Fig. 7). The two main trunks of the water vascular system extend out from the excretory bladder, one toward either side. At the posterior end of the caecum of the corresponding side the water vascular trunk turns forward and extends along the external side of the caecum, and beyond this to near the region of the pharynx. Throughout its length, but particularly in its posterior half, each tube is highly convoluted. Some of the folds lie above and some below the level of the caecum. At its anterior end the main water vascular trunk breaks up into a number of branches, which are distributed to all the tissues of the body.

The nervous system.

The brain, which consists of two lobes connected by a broad commissure, is situated just posterior to the pharyngeal pockets. It is made up of a mass of fibres among which there are scattered ganglion cells. The brain and the larger nerve trunks are surrounded by a well defined layer of nuclei which ZIEGLER (1883) took to be the ganglion cells (he mentions them only in connection with the brain). These nuclei differ very markedly from the ganglionic nuclei while, on the contrary, they differ hardly at all from the nuclei throughout the parenchyma. My own observations in regard to the nature of these nuclei are in agreement with those of SCHWARZE (1886) and TENNENT (1905) who hold that these nuclei constitute a rudimentary sheath for the nervous system. The early stages in the development of the cercaria show clearly the parenchymatous nature of these nuclei.

From the brain there are given off anteriorly four nerve trunks. These soon divide, the branches being distributed to the organs at the anterior end of the body, the anterior sucking disc and the pharyngeal pockets being particularly well supplied with nerves. Posteriorly six longitudinal trunks are given off from the brain, three on either side. These correspond in their relation and tribution to the main trunks which have been described in the a worm. In the cercaria, however, the dorsal pair of longitud nerve trunks are by far the largest. The lateral and ventral tr arise later in the development of the cercaria and up to the of encystment are scarcley more than large nerves.

Reproductive organs.

The reproductive system in the cercaria consists of well def masses of cells, which have not yet taken on the definite shape the fully formed reproductive organs. The two testes, the ov the penis sheath, the uterus and the vitellaria can be recognize sharply marked out cell areas; but not as yet as organs of sim structure to those of the adult. The testes are, in the living dition, by far the most prominent of the reproductive primor All of the others may be seen rather indistinctly.

The cystogenous glands.

In the living cercaria, the cystogenous glands appear as opaque masses, one on either side of the body, which extend ne its entire length. With a strong illumination the granular cont of the cells can be made out very clearly. In section (Fig. 7), gland is seen to be made up of a mass of large pear-shaped c each of which opens to the exterior by a separate duct. The terior of each cell is almost entirely filled with large granules w stain intensely with haematoxylin. The large nucleus is crow to one side of the cell, and the cytoplasm is restricted to the in stices between the granules of the secretion. When the cyst formed, the granular contents of these cells is thrown out and verted into the cyst, while the cells shrink to a comparatively s size. Fig. 8, a section through a cercaria which had given out cystogenous material while still within the body cavity of the re shows the condition of the gland cells after they have become from the secretion. This section shows also the thickness of mass of secretion before it has had time to harden.

In *Amphistomum subclavatum*, according to Looss, the mate from which the cyst is to be formed exists inside the gland cell the shape of "Stäbchen" which are strongly refractive and do take any stain at all.

A comparison of Fig. 6 with Looss' (1892), fig. 20, tab. 20, show clearly the striking differences between the cercariae of *Diplodiscus temporatus* and *Amphistomum subclavatum.* The shape of the body is very different. The comparative length and diameter of the tail in the two species also differs very markedly. The most fundamental difference, however, is found in the relation of the water vascular system to the tail. In *A. subclavatum* the canal leading from the excretory bladder extends nearly the entire length of the tail, near the end of which it opens to the exterior through two excretory pores, one on either side of the tail. In *D. temporatus*, as previously described, the excretory pore is on the dorsal surface of the body just anterior to the tail. Looss also figures the testes as forming a single mass which extends across the body from the internal wall of one caecum to that of the other; while in *D. temporatus* there are two distinct primordia, one for each of the testes.

4. The swarming of the cercariae.

PAGENSTECHER (1857), Looss and LANG have describe the swarming and encystment of *Amphistomum subclavatum.* These authors agree that the cercariae are born (set free from the rediae) in an early stage of development. For some time the young cercariae wander about among the organs of the host until they have completed their development, when they force their way to the surface and become free in the water. According to LANG they may escape through the digestive tract of the host; Looss maintains, on the contrary, that they reach the exterior through the respiratory cavity of the snail.

In all the specimens of infected snails that I have examined I have never found a single individual in which there were any young cercariae free in the body of the host. In the comparatively few instances where free cercariae were found they were always fully developed and ready to encyst. The only snails in which free cercariae were found were some from which mature cercariae had already been seen to escape.

In the observed instances where mature cercariae were escaping from the snails the period of liberation extended over about sixty hours. In every instance it appeared that all of the cercariae in any snail were of practically the same age, so that after a period of swarming no cercariae would be set free until a new "brood" had matured. The sectioned material, whatever was the stage in the development of the cercariae, showed that in any snail all of the sporocysts or rediae contained young cercariae which w practically the same stage of development, and which would c maturity at about the same time. The other embryos in the were always of a markedly younger stage in their developme that there would be no overlapping of the broods and a cons continuous liberation of the cercariae.

When the cercariae have been set free from the snail swim about actively for some time. Apparently the length period of activity has a definite correlation with the depth water in which the cercariae are liberated. In any event they a strong positive heliotropism, so that in a vessel into which is admitted only at the top, all of the cercariae become encyst or near, the surface of the water, on the side nearest to the of the light. The strength of the heliotropic reaction wa shown when the mature cercariae were placed in shallow glasses for study.

If the glass was left undisturbed on the table all of th cariae would become encysted on the sides of the glass near the source of light. If, on the other hand, the watch glas placed on the stage of a microscope and the light from the thrown up through the bottom of the glass, the cercariae become encysted on the bottom of the glass. By narrowin pencil of light admitted through the stage of the microscope possible to segregate the most of the cercariae in a small directly over the centre of the diaphragm, where the illumi was most intense.

Looss and Lang have stated that the cercariae of *Amphis subclavatum* may swim about for as many as fifteen to twenty hours before encysting. From eight to ten hours is given by as being the usual extent of the free swimming period. At th of this time they sink to the bottom and form their cysts.

The period of the active swimming of the cercaria of *D poratus* was always much shorter than the least period men for *Amphistomum*. None of the cercariae were observed to active for more than one hour, while in a small dish all of capable of forming a cyst would be attached within half a from the time of their liberation from the snail.

The formation of the cyst.

Under normal conditions the cercariae swim quite directly to the surface of the water and attach themselves to the sides of the containing vessel or to any floating material, such as water weeds, which may be present. When ready to begin the process of encystment, the cercariae become firmly attached by means of the ventral sucker. This attachment seems to be absolutely essential for the formation of a perfect cyst. In a small dish where there were many cercariae some of them would be unable to maintain their attachment to the bottom, and when these were separated from the substratum the extrusion of the cystogenous material would continue but a perfect cyst would never be formed.

When rediae containing nearly mature cercariae were taken from the body of a snail and kept in water, the contents of the cystogenous glands of the cercariae were extruded just as though they were free in the water. The time which would elapse before the extrusion of this material was practically the same whether the cercariae were free in the water or still contained within the body of the redia. A perfectly formed, or even completely closed cyst was never formed by any of the cercariae which had attempted to encyst while inside the redia. The cystogenous material would form a straight hollow tube about the cercaria. There were no differences in the diameter of this tube throughout its length. The tail was seldom broken off from the body. In short, the attempt to encyst resulted in the extrusion of the cyst forming material but the movements necessary for the shaping of the cyst could not be accomplished while the cercaria was unattached and the result of the attempt was the open tube.

The observations recorded in the previous paragraph suggested that the stimulus causing the extrusion of the cystogenous material was the change in the medium surrounding the cercariae. To test this point fragments of infected snail livers were examined to ascertain the stage of development of the contained cercariae. A liver containing specimens of a favorable age was divided into two parts, one of which was put in a moist chamber, while rediae teased from the other part were put into water. When the cercariae of the second lot had extruded their cystogenous material, those of the first lot were examined. None of these showed any appearence of having extruded the cystogenous material; on the contrary, most

of the cercariae were moving about within the body cavity of
redia in a perfectly normal manner.

In order to provide more nearly similar conditions the follo
experiment was also undertaken: One lot of rediae were teased
in a dish of water, those of the other lot were also separated
the liver of the snail, but were put into a liquid obtaine
crushing the livers of uninfected snails. In this experiment
results were similar to those obtained in the one last descr
The cercariae in the rediae exposed to the water extruded
cystogenous material after they had been in the water for the
time. Those inside the rediae which were put in the snail j
did not extrude their cystogenous material but finally died wi
having undergone any apparent change.

When the cercariae, after coming to the surface of the w
have become attached to some substratum the contents of
cystogenous glands begin to be poured out to the surface o
body. While this is taking place the cercaria is in constant m
The anterior end of the body bends from side to side abou
posterior sucking disc as an axis. By this means the cyst-for
material is moulded about the anterior end of the body as a
spherical mass. The posterior part of the body — behind the ve
sucker — remains comparatively quiet until the tail has been br
off by its lashing which becomes especially violent at this
When the tail has been broken off the posterior part of the
is drawn in and takes an active part in the shaping of the
The opening at the narrow end of the cyst becomes closed b
formation of a layer of cyst material after the tail has been
off and the posterior part of the body has begun its active twi
motion.

All of the cystogenous material is extruded within a short
so that the cyst is completely formed within five minutes fro
beginning of its extrusion.

When the cystogenous cells have given off their secretio
cercaria has become considerably reduced in size and now lies
up within the cyst (Fig. 5). Sparodic movements of the cer
within the cyst may continue for a number of hours after the
is fully formed. Usually the tail is carried away from the bo
its violent lashing and it may swim about for several minutes
its separation from the body.

The completed cyst is a flask-shaped structure conside flattened on one side where is was attached to the substratum. Its broader end encloses the anterior end of the cercaria. At the narrow, posterior end there is a funnel-shaped orifice where the cyst material extended up about the base of the tail before that organ was broken off.

The cyst is very fragile. It was found impossible to separate a cyst from the side of a glass jar without rupturing it and allowing the worm to escape.

The encysted cercariae were extremely sensitive to changes in the surrounding medium. When the water in a watch glass in which a number of worms had become encysted was allowed to stand for twenty four hours and was then replaced by fresh water, all of the worms came out of their cysts and moved about actively for several hours untill death ensued.

These young worms which had been in the cyst for twenty four hours differed from the cercariae in appearence in that they were comparatively much narrower, and lacked the opaque masses along the sides of the body, the cystogenous glands of the cercariae.

Some experiments were undertaken to determine the ability of the encysted worms to withstand dessication. A number of cercariae were allowed to encyst in each of several small watch crystals. When the cysts were fully formed the water was poured off from the dish and the cysts exposed to the air for varying lengths of time. An artificial gastric juice was used to dissolve the cysts after the dessication was over, and in some instances a number of the cysts were broken up with needles and the worms liberated into a dish of water. In all of the experiments a period of dessication of more than one hour proved fatal. The encysted cercariae left in water as a check for each experiment retained their vitality throughout the time of the experiment and all that was necessary in order to cause them to leave the cyst was that the water should be changed in the dish in which they were attached. The evidence gathered in another line of experiments showed that the cysts might remain in the water for at least six weeks and the young worms still be capable of recovering their activity when brought into a suitable environment.

PAGENSTECHER (1857) and REUSS (1903) have described a peculiar method of encystment for the cercaria of *Distomum duplicatum.* In this form the tail is not used at all as a swimming organ, but consists almost entirely of a gelatinnus substance When the cercaria

comes into the water the outer layers of the tail swell, and as they
increase in size push forward over the body “enclosing it as in
wall”. When the cyst has come to reach as far forward as th
ventral sucker, the animal contracts and decreases the size of th
anterior end of the body around which the cystforming materi
is extended until a complete capsule is formed. It would seem fr
Reuss' description that the cercaria becomes encysted without havi
become attached to any substratum.

Part II.

Experimental determination of the life history.

When the cercariae began to be liberated from the snails
in the laboratory, and the manner of their encystment was obse
it became apparent that some animal which obtains its foo
browsing over stones, water plants etc. along the banks of str
would probably be the next host of this parasite, whether or n
would prove to be the final one. The larvae of frogs, toads,
newts immediately suggested themselves as animals having
feeding habits.

On Oct. 30 a number of tadpoles of *Rana catesbiana* were se
and several put in each jar containing infected snails. The ta
came from a pond in the grounds of Biological Hall at the
versity of Pennsylvania, where *Goniobasis* is not found, so it s
improbable that they would be infected with the parasite
was found about Princeton. As an added precaution all o
tissues of three of the tadpoles were carefully examined w
finding parasites of any kind. When the tadpoles had been
week in the jars containing the encysted cercariae, a dead indi
was found. This one was examined for the presence of pa
with the following results: nine worms, which from the co
of the sexual organs could be recognized as young, were fo
the intestine of this tadpole. The other organs of the body
entirely free from parasites. The worms in the intestine were
2·5 mm. in length and 1 mm. in diameter at the posterior en
ot them were found in the last third of the intestine of the t
scattered throughout that part of its length. During the time
they were under examination, as was also true in every
instance, the worms remained attached to the intestine of th

by the large posterior sucker. The anterior part of the body was in almost constant motion.

Others of the tadpoles used in this experiment died from time to time and were examined for the presence of worms. The result of the examination was identical in every instance. A greater or less number of worms, ranging from ten to thirty one, was found in the posterior part of the intestine. In no case were any of the worms, or any other recognizable parasites, found in any of the other organs of the tadpoles.

The intestines of two of the tadpoles at the time of their death contained worms still within the cyst. Among the others, individuals of different ages could be recognized so it was definitely etablished that the tadpoles could serve as the host for the sexually mature worms.

Since it seemed probable from the number of worms found in each of the dead tadpoles that they had in many instances been the direct cause of the death of the host, some larger tadpoles were secured for further experiments. These were put into the jars containing encysted cercariae; but after they had remained there for a few days they were transterred to a jar in which there had been no snails. A tadpole from this jar was killed each week to note the development of the parasites. The conditions of the environment proved unfavorable for the tadpoles, and the last one of them died on Jan. 19th. 1909, after having been infected with the *Diplodiscus* for about nine weeks. The worms which were taken from the intestines of this tadpole had fully developed sexual organs, but, so far as could be determined from the examination of the jar, no eggs had been laid, or at least no embryos developed.

While the above described experiments were in progress, attempts were made to infect other animals. The intestines from infected tadpoles were fed to a turtle (*Chrysemys picta*), to toads (*Bufo lentigunosus*), and to frogs (*Rana virescens*). Two weeks after the third feeding of the turtle it was killed and the intestines examined for parasites. The only contents of the digestive tract was a dark mass in the cloaca. On microscopic examination this mass was shown to be made up of the remains of the tadpole intestines; but no trace could be found of the worms, twenty in all, which had been fed with the tadpole intestines.

The frogs, five in number, which were used in this experiment, were secured from a spring which was at least one mile from the nearest body of water in which *Goniobasis* was known to occur. Two of the number were killed on the day when the others were first fed, and their intestines examined for the presence of worms. No Trematodes were found in the digestive tract, nor in any of the other organs of the body. The three remaining frogs were fed with the intestines of the infected tadpoles from time to time, just as the latter happened to die. One of the frogs was killed a week after the first feeding. In its cloaca were found seven worms. A second frog which had been fed with the infected intestines three times, was killed three weeks after the last feeding. Five worms were found in the cloaca and in the lower part of the intestine of this specimen. The third frog was lost through its jumping out of the jar and drying up in the bottom of the can in which the jar was kept to exclude the light.

The number of worms found in the digestive tract of either of the frogs fed with the tadpole intestines was considerably smaller than the whole number of worms contained in the latter at the time of feeding. In neither case was the number in the intestines known with certainty, but in the intestines fed to the first frog twelve worms were counted. As the tadpole intestines were full of dark colored sediment and plant tissues it is probable that some of the worms were overlooked in making the count; as proved to be the case in every instance when the worms were counted in the intestine, and the latter then torn with needles to allow the worms to escape.

In discussing the transfer of the encysted cercariae of *Amphistomum subclavatum* to the vertebrate host, Looss says (1892, p. 166):

"Solche Cysten sammeln sich während des ganzen Sommers im Bodensatze der Gewässer an und werden von ihren Wirthen hauptsächlich während der Winterszeit aufgenommen. Diese verbringen, wie bekannt, die Frösche im Schlamme vergraben; sie genießen Schlamm und nehmen dabei regelmäßig auch die Wurmcysten mit auf. Bei allen den Fröschen, die ich im Winter und im allerersten Frühjahre erhielt, fand ich oft recht ansehnliche Mengen von Schlamm im Magen und Darm und in diesem bewegten sich stets einige, oft aber 20—30 ganz junge Amphistomen von den Größenverhältnissen der Cercarien: neben ihnen waren zum Teil Reste der Cysten zu sehen. Auf Grund dieser unzweideutigen Tatsachen hin habe ich es unterlassen, besondere Fütterungsversuche anzustellen.

Die Weiterentwicklung des Wurmes erfolgt, wenigstens im ersten Frühjahr, ziemlich langsam."

LANG states that, in some instances at least, the transfer of the encysted cercariae of this form comes about through the fact that the adult cercariae encyst on the skin of the Batrachians, and when this is shed, the worms are taken into the digestive tract along with the old cuticle and then find their way to the cloaca, where the remainder of their development takes place.

In the light of the observations recorded above, it seems improbable, to say the least, that the transfer of the encysted cercariae of *Diplodiscus temporatus* to the vertebrate host takes place in a similar manner. In both the bodies of water where the infected snails were found in abundance there were very few water plants of any kind floating on the surface. The banks, on the other hand, were covered with rocks which would serve as a suitable place for the attachment of the cercariae when about to encyst. On the basis of the observations made in the laboratory, it would seem that the only way in which the cysts would be likely to reach the bottom would be for them to be formed on water plants, which on their decay would set the cysts free in the water.

Granting that the cercariae become encysted on the banks, or on hard bodies floating in the water, there are two possibilities for their transfer to the digestive tract of a Batrachian where they could complete their development. They might be taken up from their place of attachment by a tadpole while it was browsing over the rocks or the vegetation along the banks. In this event it would at once be placed in its natural environment where the further development might go on to its completion. In tadpoles of *Rana catesbiana*, which may require one year to reach the adult condition, the worms might become sexually mature and give off their sexual products before the time for the metamorphosis of the tadpole.

In those tadpoles which undergo their metamorphosis during the same season in which the eggs from which they develop were laid, the fundamental changes undergone by the digestive tract of the tadpole might cause the voiding of the worms along with the old lining of the gut.

The other alternative would be that the cysts would be taken up from their attachment by some animal which could not serve for the final host, in some of whose tissues the worm would become again encysted. This intermediate host, provided it was included

in the dietary of some Batrachian, would serve as the nec
step between the encysted worm and the vertebrate host.
such a cycle in mind, many of the aquatic forms taken in the
where infected snails were abundant were examined for the en
stage of *D. temporatus.* The only animal in which cysts o
worms which might possibly be referred to this species were
was one of the common "darters", (*Etheostomata sp.*). These
fish feed continually among the rocks along the banks of the
where the infected snails abound, and can readily be taken
dip net. Nearly every one examined contained many Trema
usually of several species. Some of these worms were identi
appearence with young specimens of *D. temporatus* which had
left the cyst. These small fish are known to be taken as fo
the larger frogs, so there is thus shown a possible means
transfer.

Another possible intermediate host would be the small ta
which, after having eaten the cysts would be in turn eaten
adult frog. In this connection some observations made by
Conklin on the encysted stage of *Distomum atriventre* are instru
The vertebrate host of this Trematode is a frog, toad or
When young tadpoles were kept in the jars in which the enc
worms were abundant the latter were taken up in large nu
by the tadpoles. For some reason none of the young worms
came from the cysts remained in the gut of the tadpoles, b
of them made their way from the gut to some of the other o
of the body, where they became once more encysted. None o
organs of the body were free from the attacks of the par
They were found in the spinal canal, liver, body wall etc.
of the tadpoles when sectioned were found to contain as ma
twelve encysted worms, so they would prove a fertile sour
infection if eaten by an adult frog, as very oftens happens
natural conditions.

The tadpoles used in these experiments were the you
Rana virescens which can, in the adult stage, serve as the verte
host of *D. atriventre.* From this fact it seems probable that
must have been some reason connected with the size of the tad
which caused the worms to migrate from the gut into the
tissues. This occurence seems all the more singular when
compared with the behavior of the young worms of *Diplo*
temporatus which had been taken into the digestive tract of tad

The behavior of these last mentioned worms argues against there being any physiological difference between the digestive juices of the frog and tadpole which would account for the migration and encystment of the young worms in Prof. CONKLIN's experiments.

Part III.

Embryological.

1. The origin of the parthenogenetic eggs.

The earliest papers dealing with this question, since they were published before it had been definitely established that every cell comes from a preexisting cell, are of historic interest only. As late as the middle of the past century MOULINIÉ (1856) and PAGENSTECHER (1857) describe „endogenous cell formation" as being the mode of origin of the germ cells in the sporocyst and redia of the Malacocotylea.

WAGENER (1866) traced the origin of the germ cells to cells in the body wall of the sporocyst which become differentiated from the surrounding cells, and finally take on the characters of germ cells.

LEUCKART (1882) concluded, from his studies of the life history of the liver fluke, that the germ cells in the nurse generations may be traced directly to the egg. His idea is that they are embryonic cells which have not become at all differentiated in the formation of the sporocyst; but have come over in the body cavity of that generation.

THOMAS (1883), working on the same form which LEUCKART had studied, came to the conclusion that the germ cells may be formed either in the manner described by LEUCKART, or that then may arise from cells in the body wall.

BIEHRINGER (1884) describes their origin in the body wall.

SCHWARZE (1886) supports the view of LEUCKART: That the germ cells may be traced directly to the egg, without their having undergone any differentiation.

The later workers on this subject: LOOSS (1892), REUSS (1903), HASWELL (1904), TENNENT (1905) and ROSSBACH (1906) are agreed that the germ cells in the nurse generations arise in the body wall.

LOOSS, however, apparently considers them to be undifferentiated cells which are localized in a particular part of the sporocyst. In the summary of his paper he says: „Die Keimprodukte nehmen

bei allen 3 Formen ihre Entstehung aus einem Keimlager, w aus embryonalen, bei der Organentwicklung des Tieres nich brauchten Furchungselementen besteht (5). Dieses Keimlager bei den Embryonen eine epitheliale Auskleidung der Leibe es lokalisiert sich bei den Redien bis zur Bildung eines förm aber noch wandständigen Keimstockes, welcher bei den Cer als noch mehr individualisiert und in das Innere des Leibes g aufgefaßt werden darf.“

In *Diplodiscus temporatus* the facts seem to me to suppo opinion set forth by Thomas, namely: that the germ cells sporocyst may be traced directly to the egg, or that they arise in the body wall of the sporocyst.

A comparison of Figs. 9, 10, 11, 12 and 13, which rep sections through sporocysts of different ages, will show the g for this conclusion. In Fig. 9, which is a section through the youngest sporocysts found, the only cells present in the cavity are several eggs more than half of which are unde the maturation division. In the wall of the sporocyst the many nuclei in a homogeneous layer of cytoplasm.

In a section through an older sporocyst, as is represen Fig. 10, the body cavity contains a considerable number of practically all of which are in the same stage of developmen the whole number found in this section, only one has und the first segmentation division, while seven show a polar attached. At this stage in the development of the sporocy internal layer of the body wall is made up of a layer of cyto in which there are present a number of nuclei. This cytop layer is of practically the same thickness throughout its e The nuclei are uniformly distributed around the whole circumf of the body wall, and are of practically the same size.

In a still older sporocyst (Fig. 11). — the greater age is by the development of the contained embryos — there is show beginning of the localisation of the cytoplasm and nuclei t the ovary (Keimlager). On one side of the sporocyst the cytop layer of the body wall still retains a character similar to that in Fig. 10. On the opposite side, however, the thickness c layer has increased to more than twice that of the forme In the deepest part of the ovary each nucleus is surrounded definite mass of cytoplasm which has a sharp outline: a cell All of these cells which were cut in the proper plane sh attachment to the outer layers of the wall through a more or less attenuated strand of cytoplasm.

The earlier stages in the formation of the egg cells are shown at the edges of the ovary in the cytoplasmic layer on either side of the sporocyst. At these points, where the thickness of the cytoplasmic layer is intermediate between that of the ovary and that of the opposite pole of the sporocyst, there is taking place the formation of definite cells, by the ingrowing of cell walls which isolate a single nucleus with a definite amount of cystoplasm. At first these walls usually extend entirely through the cytoplasmic layer so that the resulting cell is roughly cuboid in shape. The distal end (next to the body cavity) soon becomes rounded and shrinks away from the inner wall of the sporocyst so that the resulting cell has the shape of a half ellipse. Further inward, toward the deepest part of the ovary, the cells become narrower at their bases and consequently higher.

In the stage shown in Fig. 12 the process of localisation has gone further. Most of the nuclei in the cytoplasmic layer are now contained in that end of the sporocyst where the cytoplasmic layer is the thickest. Some fully formed cells-(eggs) one of which has already given off its polar body, are being set free from the ovary into the body cavity; which contains embryos in two quite distinct stages of development.

In the fully developed sporocyst the ovary has the appearance which is shown in Fig. 13. Here the cytoplasmic layer about the greater part of the body cavity is very thin and contains only a few scattered nuclei. The ovary, on the other hand, is proportionately very deep. It is made up of cells each of which has its nucleus at the distal end. The oldest, tallest, cells reach nearly to the surface of the ovary and retain their connection with the inner layer of the body wall through a very thin strand of cytoplasm. In the interstices between the bases of the taller cells there are other cells which extend only a short distance from the muscular layers of the body wall. These last mentioned cells are attached by comparatively broad bases so that they are nearly hemispherical in shape. They undoubtedly represent earlier stages in the development of the eggs, comparable to those found at the borders of the ovary in the stage represented in Fig. 11.

The figure given by HASWELL (1903) of the ovary of *Echinostomum sp.* is very similar so that found in *Diplodiscus temporatus*;

although in his form the germ cells are more closely crow
together and are more regular in form, resembling a closely pac
mass of epithelial cells.

When the conditions found in a young sporocyst (Figs. 9 a
10) are compared with those present in older ones (Figs. 11 and
it becomes at once apparent that there are two definite „broo
of embryos (germ balls) formed inside the nurse generation. T
the first lot of eggs originated from cells which have come over
the embryonic development without having been attached to
body wall seems to me by far the more probable view.

As shown in Fig. 10. there are no definite cells in the cy
plasmic layer of the body wall of the sporocyst at this sta
Therefore to suppose that the eggs present in the body cavity
that stage had arisen in the wall would necessitate the infere
that cells had been formed at some places in the wall and set f
from it before any regonizable activities in the way of cell differe
tation had begun over the remainder of its area.

Another difference is shown in comparing Figs. 9 and 12.
the younger sporocyst many of the eggs in the body cavity
undergoing the maturation division. In the older sporocyst one
the eggs, still attached to the wall of the ovary, has already form
the polar body. The study of other similar instances shows that
the older sporocysts maturation commonly takes place before
eggs are set free from the ovary.

As has been pointed out by Korschelt & Heider, in th
Text Book, there is really no fundamental difference in the
cells whichever may have been the mode of their origin. The wh
question is in reality one which concerns the location, and not
all the nature, of the cells under discussion.

Reuss (1903), Tennent (1905) and Rossbach (1906) figure di
ding germ cells still enclosed in the body wall, although Tenn
states that this is of rather uncommon occurence in *Bucepha*
He says, however, that the cells which he interprets as polar bod
are usually given off before the germ cells are set free from
"Keimlager". In *Diplodiscus* I have never found a single insta
where the eggs had begun to segment before they were free in
body cavity.

Since the so-called „germ cells" in the nurse generations of
Malacocotylea prove to be parthenogenetic eggs, the "Keimlag
from which they arise must be an ovary, and on this groun

comparison with the development of the eggs in the hermaphroditic sexual generation will be of interest.

Schubmann (1905) in his study on the oogenesis of *Fasciola hepatica* finds that the oocytes become separated from the walls of the ovarium over a greater part of their surface, but that they remain for along time connected with the wall through a narrow strand of cytoplasm. His figg. 2, 3, 4, and 12, tab. 34, represent a conditions such as is commonly found in the ovary of the sporocysts of *Diplodiscus*: as is shown in Fig. 13. Concerning the function of this cytoplasmic connection he says: "Ähnlich wie bei *Ascaris megalocephala*, glaube ich, daß auch bei *Distomum hepaticum* der Eistiel die Hauptaufgabe hat, dem wachsenden Ei Nahrung zuzuführen. Diese Vermutung glaube ich stützen zu können durch die Tatsache, daß ich im Eistiel stets dunkle, körnige, unregelmäßige Partikel sah, die sich dort, wo der Stiel in die Oocyte übergeht, besonders zahlreich finden. Die Partikel sind der Eizelle durch den Stiel zugeführte Nahrungsmassen. In den figg. 2, 3, 4 sieht man deutlich, wie diese dunkler gefärbten Körperchen in die Zelle hineinrücken, um von dieser als Nahrung aufgebraucht zu werden."

Kerbert (1881) describes the same appearence of the oocytes in *Distomum westermanni*, but gave to it a very different interpretation. He considered the pedicle to be a long thin amoeboid process of the egg cell. He makes no mention of any special function for this part of the cell and apparently considered its relation to the wall of the ovarium to be simply contact instead of cytoplasmic continuity.

The Maturation of the Parthenogenetic Eggs.

Grobben (1882), Leuckart (1882) and Schwarze (1886) held that the germ cells within the nurse generations of the Malacocotylea were true parthenogenetic eggs, and that consequently the life cycle was heterogenesis.

Biehringer (1884) characterized the development as "internal budding". Schauinsland (1883) described the developmental cycle as a true alternation of generations, to which he gave the special name Alloiogenesis.

Looss (1892) characterized the development as a metamorphosis, he says: "Die Entwicklung aller Formen kann ohne Zwang aufgefaßt werden als eine Metamorphose, welche auf mehrere Generationen sich verteilt (Sars, Leuckart). Der Annahme einer Parthenogenesis scheint mir der Umstand entgegen zu stehen, daß aus d Keimstocke nicht nur Eizellen, sondern auch andere Elemente (War zellen) gebildet werden."

None of these investigators gave any attention to the questic of the formation of polar bodies. Their arguments for or against parthenogenetic development of the germ cells are purely theoretic

In recent years a number of papers have appeared which de with the question of the character of the development of the ger cells in the nurse generations, although none of them have be strictly cytological in character.

Coe (1896) in studying *Fasciola* (*Distomum*) *hepatica*, — the for on which Leuckart had based his conclusions concerning parthen genesis in the Malacocotylea — was unable to find any maturati divisions or polar bodies. He states, however, that the absence the formation of a polar body i. e. the division of a germ cell in two unequal cells only one of which develops further, does n necessarily weigh against the „Einatur" of the germ cell. H would trace back the maturation process into the earlier history the germ cells where at some definite division a parent cell divid into two cells, each of which is a definitive egg. He compares th process to the development of spermatozoa, where all of the ultima cells become of the same size and character. This conclusion ha of course, no observations for its support, and besides it is oppos to all the known facts of oogenesis.

Reuss (1903) concluded as the result of his studies on *Distomu duplicatum*, that there was a true maturation of the germ cel within the sporocyst of this species. His evidence was, howeve very fragmentary, consisting only in the fact that he found sma cells with coarsely granular, apparently degerating, nuclei attache to the larger germ cells, before these began to segment. He figur (fig. 12 and 13) what he considers to be a maturation divisio The only evidence in favor of this conclusion is, however, that the is no small granular nucleus attached to the germ cell in which t spindle occurs. This fact appears to be for him sufficient eviden that the result of the mitosis figured would have been tbe cutti off of a polar body — (one of the coarsely granular nuclei) — fro the large germ cell.

Reuss figures three polar bodies as being the number usuall found. He states that they persist for a long time attached to t egg; and it would seem from his figures of some of the later se mentation stages that he had counted the so-called "polar bodies" as segmentation cells.

HASWELL (1903) noted the same sort of cells, "with deeply staining homogeneous nuclei", lying near the ovary "(Keimlager) in the body cavity of the sporocyst of *Echinostomum sp.* He says in regard to these cells: "If they are not to be regarded as of the nature of polar bodies it seems difficult to account for them".

TENNENT (1906), in describing the development of the germ cells in *Bucephalus* says: "The first appearence of activity in the germ cell itself is the cutting off of a small cell (fig. 15, b.) (As has been already noted this may take place in the wall.) This cell differs in appearence from the much larger germ cell in that its nuclear contents are much more coarsely granular. These contents in a later stage are seen gathered together into two masses, the larger nucleus of the germ cell having in the mean time moved to the opposite periphery of the cell. In stages which I believe to be still older, a second cell has made its appearence, and still later there are three, two of which seem to be the result of the division of the first cell (fig. 15, c.)."

ROSSBACH (1906) points out the resemblance of the so-called polar bodies of REUSS to the very young germ cells. He states that he has never seen the formation of a polar body in any of the material which he has studied, and expresses a doubt as to the occurence of this phenomona in any of the eggs (germ cells) arising in the nurse generations of Trematodes. He admits, however that there is a possibility that maturation of the germ cells may occur. "Obwohl es mir, gleich wie COE am Miracidium, nicht geglückt ist, bei Sporocysten und Redien Richtungsspindeln festzustellen, so will ich damit jedoch nicht behaupten, daß eine Reifung der Keimzellen nun auch ausgeschlossen sei. Die Keimzellen haben ja ohne weiteres eine große Ähnlichkeit mit Eizellen, neuerdings sind auch solche Reifungserscheinungen von den Keimzellen der Dicyemiden, wo die Verhältnisse sehr ähnlich liegen, durch HARTMANN bekannt geworden. Alles das spricht dafür, daß vielleicht auch die Keimzellen der Ammen der digenetischen Trematoden eine solche vor ihrer Furchung durchzumachen haben; wenn es mir selbst auch nicht gelang, eine solche nachzuweisen, so mögen andere Forscher darin vielleicht glücklicher sein! . . . Solange nicht gezeigt wird, daß diese Zellen aus der Keimzelle vermittels einer Reifungsspindel hervorgehen, ist es mir unmöglich die REUSS'sche Auffassung anzuerkennen."

There are often found, according to Rossbach, germ cells still within the keimlager near which are situated a number of smaller cells with coarsely granular nuclear contents. These smaller cells are said by him to be set free into the body cavity of the sporocyst along with the germ cells with which they remain in contact. These are, he says, the same sort of cells as those which have been described by Reuss as having arisen from the germ cells by maturation divisions.

As regards the nature of the development of the germ cells, Rossbach supports and elaborates the view of Coe, that the formation of a polar body is not of necessity the criterion for determining the "Einatur" of the germ cells in the nurse generations of the Malacocotylea.

He would, with Coe, trace back the maturation („ursprüngliche Richtungsteilung") to the division of a primitive germ cell into two functional egg cells, each of which would on its division give rise to a germ ball.

In order to provide a basis for the comparison of the details of the maturation division it has seemed advisable to describe the process in a typical cell of a germ ball. In any resting cell the nucleus is comparatively very large and is usually centrally placed in the cell (Fig. 14). In all stages, except in the egg and in the first two or three divisions in the formation of the embryo, the cell walls are rather indistinct, and often cannot be made out at all.

All the nuclear divisions, both those in maturation and in the segmentation are entirely intranuclear. In no stage in mitosis, so far as I have been able to make out, is there an opening of appreciable size between the interior of the nucleus and the cytoplasm of the cell body.

Within the nucleus practically all of the chromatin is gathered into one (sometimes two) caryosomes. These structures are surrounded by a mass of less densely staining material of which narrow strands stretch out for some distance toward the periphery of the nuclear membrane. The remainder of the contents of the nucleus are quite homogeneous and stain very lightly. The caryosome is almost always situated a little excentrically in the nucleus, but there seems to be no constant relation between its position and any other factor such as the cleavage planes, shape of the nucleus, etc.

As the mitosis is intranuclear I have given considerable time to searching for the centrosomes in the resting cells. In the telophase of mitosis the centrosomes for so long as they remain clearly distinguishable are situated near to the mass of chromosomes which are fusing to form the caryosome. In the latest telophase, after the caryosome has become a sharply marked out mass and is surrounded by the layer of less densely staining material, the centrosomes can no longer be distinguished. The most careful study has failed to show whether the centrosome in the resting stage is situated within the darkly staining material about the caryosome, or whether it lies within an indentation of the caryosome itself so that it would be within the circumference of the latter body. The centrosomes first become apparent in the division of a nucleus at a stage when the spireme formation is well advanced. At this time there are two centrosomes and a well developed spindle between them. All of the earlier stages in the development of the achromatic figure are concealed within the caryosome, or the darkly staining mass about it.

The Formation of the Spireme.

The nuclei in the following series of figures were drawn from different embryos, which accouts for the differences in their sizes. The resting cells, as has been previously described, have the appearence shown in Fig. 14. The first stage in the prophase of mitosis shows the beginning of an increase in the size of the caryosome at the expense of the nucleoplasm, (Fig. 15). As the nucleoplasm is taken up by the caryosome there become apparent a number of threads, (linin?) which connect the central mass with the nuclear membrane, Between these strands there are left empty spaces, (Figs. 16 and 17), so that the central mass appears to be suspended by threads from the nuclear wall.

In the formation of the “suspensory threads” it can be determined that each one is double, having been formed by the apposition of the outer walls of an area of nucleoplasm, (Fig. 15). In early prophase stages the contents of the nucleus, outside of the caryosome, appears to be marked off into sectors by denser areas which later go to form the “suspensory strands”. In the resting nucleus, however, I have never been able to distinguish any differentation in the nucleoplasm which could be interpreted as marking out definite areas.

At a stage when the greater part of the contents of the nucleus has been taken up by the caryosome, (Fig. 16), the latter body has lost its intense staining reaction and now has a uniform homogeneous structure, and stains a comparatively light color.

The next stage in the formation of the spireme is marked by

the appearence of rather fine, densely staining, granules which a
scattered throughout the “central mass”, (Fig. 17). These granu
increase in size, (Fig. 18), until they fill the central mass. Whe
the granules come in contact they fuse with one another, to fo
a continuous thread, (Figs. 18, 19 and 20). When they first beco
arranged to form the spireme thread the latter lies coiled up with
the central mass, which has not as yet increased in size since t
time of the condensation of the contents of the nucleus.

When the spireme becomes definitely established as a threa
there ensues a period of rearrangement of the nuclear contents. T
spireme thread grows at first faster than the diffusion of the “centr
mass” takes place so that at a number of points the chromatic thre
forms protuberances on the surface of the latter, (Fig. 19). In lat
stages the rearrangement of the less chromatic part of the nucle
contents goes ahead of the growth of the spireme thread, as show
in Fig. 20. This rearrangement of the nucleoplasm follows apparent
the same lines, traversed in an opposite direction, when it was tak
up by the caryosome. Each “suspensory strand” becomes separat
into its two component parts, (Figs. 18 and 19), and these wa
become further and further separated with the expansion of t
“central mass”. In a stage as shown in Fig. 20, the nuclear conten
have become extended to fill the entire cavity inside the nucle
membrane, while there is yet a more darkly staining area abo
the spireme thread. In later stages when the chromosomes ha
become differentiated, and are being arranged to form the equatori
plate, the whole of the nucleoplasm has a uniformly light staini
reaction.

The spireme thread in its later growth comes to fill the enti
nucleus (Fig. 23). Sometimes before the redistribution of t
nucleoplasm is completed, the spireme thread becomes constricted
a number of points so that it appears like a chain of very lo
beads. The characteristic shape of the component granules (Fig
24 and 25) serves to distinguish such a stage from one in whic
the spireme thread is being formed by the fusion of the granul
as in Fig. 17.

The constrictions separating the chromosomes become deep
until the latter are sharply marked off from one another, (Fig. 2
Later the chromosomes become separated from one another by
little distance, but for some time they remain connected together
thin strands of achromatic material (Fig. 25).

In a side view of a nucleus which seems to be in a stage slightly earlier than the last figured, the chromosomes appear massed together while they are evidently being arranged to form the equatorial plate. In the metaphase the chromosomes have lost their connecting fibres, so that each one is distinct from all the others (Fig. 26).

Fig. 27 and 28 represent later stages in the formation of the chromosomes and their arrangement about the middle of the spindle to form the equatorial plate. In these figures it will be seen that the centrosomes are very prominent. They are comparatively large and stain, with the haematoxylin, practically as deeply as the chromosomes.

In Fig. 26, which represents a section through the equatorial plate of a nucleus in the metaphase of mitosis, the chromosomes are seen to be arranged throughout the greater part of the area of the nucleus. In other sections they were found in a ring about the periphery of the nucleus, so there is no constancy in their arrangement.

In such a section, at right angles to the spindle, the chromosomes are usually somewhat dumb bell-shaped, and of practically equal size. In all cases where the section is cut in such a manner as to show the entire number of chromosomes, there are found to be sixteen. In especially favorable side views of the mitotic figure, before the splitting of the chromosomes, this count can be verified (Fig. 28).

In side views the shape of the chromosomes is elliptical, their diameter being about one half their length. At no time in the mitosis do the chromosomes assume the long filamentous shape such as has been figured by GOLDSCHMIDT (1905) for the chromosomes in the egg from the hermaphroditic generation of one of the Heterocotylea (*Polystomum*).

In the figure last mentioned, as well as in all figures of side views of the mitotic figure, the prominence of the achromatic figure is well shown. In reality the linin fibres are much stouter than they are shown in any of the figures. They are represented by thin lines only on account of the method by which the drawings were made. If they had been drawn to scale they would, in a black and white figure, be nearly as prominent as the chromosomes. Each of the spindle fibres is made up of a number of elements. There were most commonly four of these elements in each fibre.

To give an idea of their diameter it may be said that in any
each fibre was of about the same diameter as the centrosome
the same nucleus.

In the anaphase of the division the chromosomes are dr
apart, from the equatorial plate, each daughter plate converg
toward the centrosome at the corresponding side of the nucleus.
the beginning of this process the nucleus still retains its ne
spherical shape; but in later stages, when the chromosomes h
been drawn up close to the centrosomes, the outline of the nuc
becomes elliptical. The long axis of the ellipse corresponds with
of the mitotic figure.

At about this stage (when the daughter chromosomes
gathered in a mass near the centrosomes, and those in the ce
of the mass have begun to lose their identity) there is the beginn
of a constriction about the middle of the nucleus. This constric
seems to take place rather slowly, to judge from the correla
between the stages in the constriction of the nucleus and
condition of the mass of daughter chromosomes found in each of its e

On one occasion a division was observed to take place
living sporocyst which I was studying. When this nucleus
first observed (it is impossible to state with certainty whethe
not it was a germ cell, although its location at the broad en
the sporocyst as well as its large size would make this infer
seem probable) it was spherical in shape, and differed in
appreciable respect from the nuclei surrounding it. When not
again it had become considerably elongated and there was a sl
constriction about its middle part. For the remainder of the
until the complete separation of the daughter nuclei, this nuc
was constantly under observation. My notes show that from
time when it was first noticed that this nucleus was different
the others, until the time when the daughter nuclei had separa
a period of twenty minutes had elapsed.

As seen under the comparatively low magnification used,
that could be observed was that the constriction became gradu
deeper and deeper until finally the nucleus became separated
two parts. The whole cycle of events resembled an amitotic divi
which, indeed, at the time I considered it to be.

The study of sections of preserved material has shown, howe
that the facts above recorded are of normal occurence during
mitosis in this trematode.

The constriction about the middle of the nucleus becomes deeper and deeper involving, apparently, only a short section of the nuclear membrane. In the last stages of the separation of the daughter nuclei they are connected only by a narrow strand of the membrane, and each half is nearly spherical in shape. When the two sides of the nuclear membrane come together, at the end of the period of constriction, they fuse and the connecting strand of tissue is broken.

At no time, so far as I have been able to determine, is there an open communication of any appreciable size between the cytoplasm of the cell body and the interior of the nucleus.

After the daughter nuclei have separated from one another they move to opposite ends of the cell and then the cytoplasm of the body of the parent cell becomes separated into two parts by the ingrowth of a constriction of the cell wall.

Within the daughter nuclei the chromosomes continue to fuse into a mass until all of them have lost their individuality and they form a body with a regular outline, which is the caryosome. The less densely staining material becomes localized about the caryosome, and we have again a typical resting nucleus of a cell of a germ ball.

The striking similarity between the late telophase in the mitosis of *Diplodiscus* and a typical amitotic division suggests a possible explanation of the results which have been obtained by some of the investigators of cell division in Cestodes and Trematodes. In a stage such as is shown in Fig. 30, and as would appear in all later stages of division, we have an appearence which might easily be interpreted as a true amitotic division, since practically all of the evidences of mitosis have dissappeared. The later telophase would present conditions showing all of the usual stages of amitosis, such as have been figured by CHILD (1905) for the various tissues of *Moniezia expansa.*

In all of my preparations of *Diplodiscus*, while there have been many instances of what at first sight seemed to be amitosis, a careful study has shown that there would invariably be some of the characteristics of mitosis by which it could be definitely determined that the division of the nucleus under observation had taken place by the usual method of mitosis as previously described.

The maturation division.

The maturation division in the parthenogenetic eggs differs in several respects from a typical segmentation division, as above described.

The resting nucleus in the oocyte is similar to that in one the segmentation cells. In the maturation stages, however, ev before the formation of the equatorial plate, the nucleus becon elongated in a direction corresponding to the long axis of the e In every observed instance where the maturation division v taking place, one end of the nucleus was close to the wall of cell, and, indeed, usually in contact with it. In the eggs s attached to the ovary the nucleus is apposed to the cell wall the broad, distal, end of the egg. In those eggs in the you sporocyst which were undergoing their maturation while free in body cavity, there was, of course, nothing by which any defin orientation could be determined. In all cases, however the sha of the egg was elliptical instead of nearly spherical as in t segmentation cells.

The formation of the spireme for the maturation division diff very markedly in one respect from the same process in one of t segmentation nuclei. In the maturation division I have never fou a stage corresponding to that in the segmentation division, whe the greater part of the contents of the nucleus are gathered into a single central mass which is “suspended” from the nucle membrane.

In the maturation division, on the other hand, the caryoson although it increases slightly in size, never becomes a light staining mass. While granules can be recognized in the caryoson after it has reached its greatest size for this division, they are fr the first, larger and less distinct than in the segmentation nucle They immediately fuse to form the spireme thread, which lies the densely staining mass of nucleoplasm, that formerly surround the caryosome.

On the growth of the spireme this densely staining area increas in size, and becomes lighter in color until it is finally indistinguis able from the remainder of the nucleoplasm.

The fully developed spireme in the maturation division occupi a smaller portion of the nucleus than does the spireme in t segmentation divisions. It is always confined to the portion of t nucleus nearest the cell wall of the egg (Fig. 32). The chromosom are formed from the spireme in the manner already described f the segmentation divisions (Fig. 33).

In Fig. 36 and 37, which are drawings from eggs undergoin their maturation free in the body cavity of the sporocyst t mitotic figure shows several points of difference from a common segmentation mitosis. In some instances (Fig. 37) the spindle extends through nearly the entire length of the nucleus. In most instances, however, the mitotic figure occupies only a comparatively small part of the nucleus (Fig. 36).

The chromosomes in the maturation division are very different in appearence from those seen in the segmentation stages. On comparing Fig. 35, where the chromosomes are arranged in an equatorial plate for the maturation division, with Fig. 45, a section through the plate of the first segmentation division, the difference in the size and shape of the two sorts of chromosomes is strikingly shown. In any of the maturation divisions the size of the chromosomes is scarcely more than one half that of those present in a segmentation division. It should, however, be pointed out, when such a comparison is made, that in segmentation mitoses there are great differences in the size, and to a lesser extent in the shape, in the chromosomes found in different nuclei, even those within the same germ ball.

In the shape of the chromosomes during maturation there is as striking a difference as was noted in their size. In a polar view of the equatorial plate of a maturation mitosis (Fig. 35), the chromosomes are nearly circular in outline. In a side view of the spindle (Fig. 34), they are somewhat elongated in the plane of the plate; but this elongation is very slight when compared with the chromosomes in a segmentation mitosis as is shown in Figs. 26 and 45.

The achromatic part of the mitotic figure is made up of finer fibres in the maturation than in the segmentation division. In some eggs it can be only imperfectly made out. This may in part be due to the fact that the contents of both the egg cell and its nucleus are denser than are those of the segmentation cells of the embryo.

The centrosomes alone, of all the structures within the nucleus, appear during mitosis to be of the same size and character in both the maturing eggs and in the segmenting cells of the embryo. The location of one centrosome within the nucleus at the time of the maturation division is often some distance from the corresponding end of the latter; but otherwise there is apparently, so far as concerns the centrosomes, no difference between the two types of division.

In the anaphase of the maturation divisions the daughter
are drawn apart so that the one which is to remain in the n
comes to lie in the centre of that body; while the one which
be extruded into the polar body goes to the extreme end
nucleus where the latter is in apposition to the cell wall.
earliest stages in the extrusion of a polar body I have been
to find in any of my material. In a later stage as shown in I
the interior of the polar body is continuous with that of the n
through a rather broad stalk. In this stage the chromosor
both of the daughter plates still retain their individuality
indeed, have not been drawn up to form a close cluster, so it
probable that in this instance we have to do with a compara
early stage in the process.

In the egg represented in Fig. 39, there is still a very n
connection between the polar body and the nucleus. The chromo
of each daughter plate have, for the most part, lost their ide
They form a homogeneous mass which shows only about the
border any trace of its origin from separate chromosomes.
the evidence given in the last mentioned figure it would seem tha
as in the segmentation divisions, the constriction of the nucle
cut off the polar body is a comparatively slow process.
separation of the polar body becomes complete only afte
chromosomes in both daughter plates have become indistinguis
from one another. In the nucleus of the ripened egg they
fused to form the caryosome; in the polar body, on the other
there is never a sharply defined body comparable to the cary
but the chromatin material becomes scattered throughou
nucleoplasm in the form of irregular granules. At first
granules are distinct from one another. Later the whole m
the polar body has a uniform staining reaction, as if the chro
material was evenly distributed, as in a solution.

At no time in the formation of the polar body, at least
stage later than the earliest one that I have found (Fig. 3
there any evidence that the polar body contains other than n
material. In Figs. 38 and 39 it will be seen that the mem
which forms the wall of the polar body is a direct continuat
the nuclear membrane. It seems probable, also, that the sepa
of the polar body is accomplished in the same manner as t
the daughter nuclei in a segmentation cell: i. e. by the fus
the nuclear membrane when the two sides come together,

close of the period of constriction, and the subsequent breaking of the strand of tissue formed by their fusion. From the appearence of the cell wall as is shown in Fig. 38, it would seem that there was an active pushing out of the nucleus at the time of the extrusion of the polar body. In this egg the cell wall extends up around the narrowest part of the constriction between the nucleus and the polar body, just as would be true for any elastic membrane which had been ruptured by pressure from within.

In only a single instance among all the eggs with polar bodies found among my material, was there any evidence of the division of a polar body after its separation from the egg. In the egg shown in Fig. 41, there are two small bodies attached, both of which have the characteristic appearence of polar bodies. There is also in favor of the conclusion that these bodies came from the division of a single one the fact that taken together they make a mass about the size of an ordinary polar body.

A comparison of the several figures of eggs with polar bodies, shows that there are considerable differences between the sizes of the latter. If my interpretation of the method of the extrusion of the polar bodies is correct, the difference in their size is not surprising. Prof. CONKLIN, in a paper as yet unpublished, points out the fact that in the eggs of *Crepidula* the size of the polar bodies can be controlled by differences in pressure. Within the sporocysts, or rediae, of the Malacocotylea while they are within the body of the host, there must be considerable variations in the pressure from time to time, so in the light of the results just quoted, this fact might have some considerable influence in determining the size of the polar body cut off from any egg.

The maturation phenomena just described conform to the type generally occurring among parthenogenetic eggs from which only one polar body is given off. There is no numerical reduction of the chromosomes. In the maturation division the chromosomes are halved, just as in a segmentation division, in other words the division is purely quantitative.

2. The segmentation of the egg and the formation of the germ layers.

The nuclear phenomena concerned in segmentation have been described in detail in an earlier section of this paper and need not be discussed in this connection.

In the first segmentation division (Fig. 44), the spindle becomes excentric in position so that the equatorial plate lies to one side of the chief axis of the egg. As the result of this division there are formed two small cells of unequal size. The diameter of the larger one exceeds that of the smaller one by an amount nearly equal to the diameter of the polar body. At the second division another small cell is cut off from the larger one. There are thus formed two small cells (primary micromeres) and one large cell (macromere) (Fig. 47 In the next few divisions the micromeres alone take part, while th macromere remains in a resting condition until it has been almos completely overgrown by the cap of micromeres. The division o the micromeres usually takes place simultaneously, so that a stag in which there are five cells (Fig. 49) follows one in which the are three. In some instances, however, the sections show embry made up of four cells as in Fig. 48.

Up to this stage in the segmentation the outlines of the ce remain tolerably distinct, so that the endoderm cell (macromere) c clearly distinguished from the ectoderm cells (micromeres) by size. In later stages the outlines of the cells become lost so th the whole embryo makes up a syncytium. In many instances, ev after the cell outlines have dissappeared, a line separating the ce which have come from the primary macromere (endoderm) fr the enclosing mass of cells (ectoderm) remains distinct up to time when the endoderm cells become arranged to form the prim dium of the gut (Figs. 53, 54 and 56).

The formation of the investing membrane.

When the embryo has reached the nine cell stage two of micromeres become distinguishable from the others by the m hyaline appearence of their nuclei. These two cells are pushed from among the others so that they lie on the surface of the m of cells. Their cytoplasm becomes flattened out to form an exceedin thin layer over the surface of the remaining cells. The invest cells divide once after they have come to lie on the surface of embryo, so that when completed the investing membrane cons of four cells. The nuclei of these cells remain for some time prominent protuberances on the surface of the embryo (Fig. Later they become flattened out so that they are hardly more t twice the thickness of the cytoplasmic part of the membrane. U mately they seem to disappear entirely, as sections through l stages of the embryo, after it has begun to elongate, fail to show any traces of these nuclei.

SCHWARZE (1886), TENNENT (1905) and others, have described the origin of the investing membrane of the germ balls as taking place in the manner above described.

ORTMANN (1908), in describing the early germ balls in the miracidium of *Fasciola hepatica*, says: "Bereits in diesem Stadium sah ich den Keimballen eine Art Hüllzelle aufsitzend, die kapuzenartig ihn überwölbte, wie ich dies schon oben bei Besprechung der Hüllmembran angab (tab. 14, fig. 35 *Klz*). Daß sie ein Blastomer des Keimballens darstellt, scheint nach tab. 14, fig. 35, 36a, b, 37 sehr wahrscheinlich. Wir finden eine oder zwei solcher kallottenförmiger Zellen auch noch in weit spätern Stadien dem Keimballen aufsitzen, ohne daß eine weitere Differenzierung an ihnen vorgegangen zu sein scheint. Ihre Ausbildung zur Cuticula, wie sie ROSSBACH beschreibt, war nie zu beobachten. Offenbar findet sie noch nicht statt, solange das Miracidium noch im Ei sich befindet oder freilebend ist, sondern erst, wenn es in seinen ersten Wirt gelangt ist."

His figures of these young germ balls (fig. 35, 36a, 36b and 37, tab. 14), when compared with the germ balls represented inside the body cavity of the miracidium (his fig. 38, tab. 14) show clearly that in the cells(?) attached to these young embryos we have to do with quite different structures from the investing cells of the older embryos. The bodies attached to the two celled embryos (contrary to all other recorded instances the cell outlines are less distinct in the younger stages) contain no cytoplasm at all. They are clearly shown as being made up of a small nucleus-like structure covered by a thin membrane, so it seems hardly possible that from these alone are to come the cytoplasmic membranes which will enclose the embryos.

In the light of my own observations on the formation of the polar bodies of the parthenogenetic eggs of *Diplodiscus*, it would seem most probable that the nuclei figured by ORTMANN represent polar bodies which are still attached to the segmenting eggs. The structure of the attached nucleus in fig. 36b, lends suppord to this view. In this figure the nucleus is represented as containing a mass of chromatin which consists of rather fine granules scattered irregularly through the interior of the nucleus, just as is the appearance in early stages in the degeneration of a polar body.

In the same paper the formation of the "Hüllmembran" in embryos from fertilized eggs is, on the other hand, described as taking

place through the activity of certain of the segmentation cells. A
the segmentation has gone on for for some time, so that gastrula
is nearly completed, a few of the nuclei in the ectoderm bec
distinguishable from the others through their smaller chromatin
tent and clearer appearence. At first they form a distinct l
about the ectoderm (the segmenting egg is in this stage surrou
by a mass of yolk cells), later the "Hüllmembran cells" mig
through the yolk and form a layer the over surface of the la
which is thus brought inside the body of the embryo.

From the above account it is apparent that in the only s
(the development of the embryo from the fertilized egg) in w
the development was studied, the formation of the investing m
brane took place in the usual manner. So it seems all the m
improbable that in the same species two different methods of
formation of the same organ would be found.

In the Heterocotylea, according to Halkin (1902), Goldschi
(1902, 1905) and Kathariner (1904), the enveloping membrane a
in an entirely different manner.

In *Zoogonus mirus*, according to Goldschmidt, the egg cap
contains an egg cell and two "primitive yolk cells", which h
come from the vitellarium. These last mentioned cells are not
cells which function only by giving nutriment to the embryo;
on the contrary, from them is formed the investing membrane a
the developing embryo. These cells have the power of divi
once, so that the membrane is composed of four cells, just as in
embryos from the parthenogenetic eggs in the Malacocotylea.

In the general part of his paper Goldschmidt (1905) discu
the relationship of the investing membrane and the ectoderm
trematodes. He reaches the conclusion that in embryos from f
lized eggs the "Hüllmembran" is never formed from segmenta
cells. It is formed either by the rearrangement of the yolk nu
(nuclei of the yolk cells), as in *Monostomum*, or by the sprea
over the embryo of the "rudimentary yolk cells", as in *Zoogo*
The so called "primary ectoderm" of the older writers is thus
a part of the embryo at all.

The casting off of the secondary ectoderm, as described
Schauinsland for *Distomum cylindraceum* and *D. mentulatum*, G
schmidt characterizes as inconcevable, since in most miracidia,
body cavity lies directly under the ectoderm.

In the development of the embryos (germ balls) from parth genetic eggs of the Malacocotylea the investing membrane is, in all known instances, formed from two of the micromeres ot an early stage in the segmentation. In *Diplodiscus*, and in other forms where the early segmentation has been described, it appears that the second cleavage separates two cells which are to give rise to the ectoderm from one cell which is to give rise to the endoderm and mesoderm. The cells which go to form the investing membrane are thus clearly ectodermal cells which in an early stage in the development of the embryo become set apart for the performance of a special function, as a protective and osmotic membrane.

That these four investing cells represent the entire ectoderm, as has been maintained by Rossbach, cannot, in the light of our knowledge of the segmentation of both the fertilized and parthenogenetic eggs of trematodes, be admitted. Another assumption of this theory, i. e. that all of the cells between the primordium of the gut and the investing membrane constitute the mesoderm, violates a firmly established principle of comparative embryology, in that it necessitates the origin of the central nervous system and the epidermal sensory organs from the mesoderm.

In the embryonic development of many other animals, particularly the worms and molluscs, certain cells among the micromeres become early set apart for the formation of larval organs, which in the further development of the embryo cease to be functional and are either resorbed or cast off. In the investing membrane of the embryo from the parthenogenetic eggs of Malacocotylea we have, in my opinion, an exactly parallel case, and one, therefore, which in no way concerns the question of the formation of the germ layers.

According to the observations of Tennent (1905) and Rossbach (1906) with which my own agree, the investing membrane persists about the embryo until the formation of the permanent cuticle of the cercaria takes place. On the formation of the last mentioned structure the investing membrane is no longer useful and it becomes buried in the secretion from the cuticular glands, probably becoming a part of the permanent cuticle, or perhaps forming the basement membrane.

In considering the development of the embryos from parthenogenetic eggs (germ balls) few investigators have given any special attention to the formation of the germ layers. Thomas (1883) describes a true invagination as taking place in the germ balls of *Fasciola hepatica*, which are to form rediae.

Schwarze (1886), describes the early development of the cerca in the following words: "Aus jedem der soeben beschriebenen Ke ballen geht durch unregelmäßige Klüftung eine Morula hervor, de einzelne Zellkerne nicht die Größe des primären Zellkerns erreic im übrigen aber denselben durchaus ähnlich sehen. Zellgrenzen s in dem spärlichen Plasma nicht wahrzunehmen. Diese primä Klüftungsprodukte, aus welchen alle übrigen Gewebe hervorge bezeichne ich als Urparenchym oder Meristemzellen (fig. 2 *me*).

Schon sehr bald bemerkt man an diesen Keimkugeln wei Differenzierungen. Zunächst treten an verschiedenen Stellen, ha sächlich aber im Centrum derselben, Zellkerne auf, welche sich denen der Meristemzellen auffallend unterscheiden (fig. 2 *gz*)."

Tennent makes no mention of the formation of the germ la although his figures and description of the early stages in the mentation show that a single large cell (macromere) is overg by the cells arising through the segmentation of a single pri ectoderm cell (micromere).

Rossbach (1906) in his description of the development o redia places the differentation of the germ layers in a comparat late stage in the development, after the embryo is made u many cells and has become elongated. He says, p. 403: "Be zunehmenden Längsstreckung des Keimballens jedoch tritt nun in seinem Innern eine große Veränderung auf, indem in s Längsachse sich eine Zellmasse zu konsolidieren beginnt: die lage des Entoderms. Durch diesen Vorgang wird die bisher nicht differenzierte Masse des Keimballens in eine zentrale axiale und zugleich in eine periphere gesondert. Durch diesen wicklungsprozeß sind nun auch die 3 Keimblätter der Redie standen: der axiale Zellstrang als Entoderm und das zwi diesem und der Cuticula, die, wie wir an ihrer Entwicklung s das Ectoderm repräsentiert, gelegene Mesoderm, aus welchem let sich nachher die Keimzellen, das Exkretionsgefäßsystem, die drüsen und das Nervensystem entwickeln."

The earliest embryo figured by Rossbach consists of at twenty seven cells (twenty seven nuclei are represented in wh states to be an optical section of the germ ball) and he mak mention of the earlier segmentation stages.

In the development of the fertilized eggs, on the other ha number of investigators have described a true gastrulation.

Zeller (1876) describes for *Polystomum integerrimum*, a course of development which is clearly a case of epibolic gastrulation.

Schauinsland (1883), on whose description of the development in several species of trematodes most text book descriptions are based, recognized the formation of a morula, but could distinguish no trace of gastrulation or formation of germ layers.

Within recent years, a true gastrulation has been described in the development of several trematodes.

Halkin (1901) and **Goldschmidt** (1902) describe the gastrulation of *Polystomum*. **Kathariner** (1904) finds essentally the same process taking place in *Gyrodactylus elegans*.

Goldschmidt (1905) describes a very unusual method of formation of the germ layers in *Zoogonus mirus*. In this specis there is cut off from one cell of the twelve celled morula, by "perikline Teilung" a cell: "die nunmehr das Zentrum des Embryos einnimmt und als primäre Entodermzelle anzusprechen ist. Indem diese primäre Entodermzelle sich in zwei hintereinanderliegende Zellen teilt, ist die Bildung der primären Keimblätter vollzogen. Ein aus 2 Zellen bestehendes Entoderm wird von einer Schicht kubischer Ectodermyellen umgeben, die sich ihrer Größe und ihrem Bau nach als verschiedenwertig erweisen."

Ortmann (1908) describes the usual gastrulation as taking place in the development of *Fasciola hepatica*. In his figures of the later stages of segmentation, before the etablishment of the primordia of the organs of the miracidium, all cell outlines become obliterated so that the arrangement of the layers cannot be made out.

In *Diplodiscus temporatus* the endoderm cells retain their identity for some time after all cell walls have dissappeared in the ectoderm. Even after the outlines of the endoderm cells have been lost, the line of demarcation between the entoderm and ectoderm remains clearly distinguishable for some time (fig. 53). In some of the embryos the cells making up the endoderm remain distinctly marked off from the other cells throughout the segmentation, and until the characteristic arrangement of the endoderm to form the primordium of the gut has taken place. In the greater number of the embryos, however, the distinction between the layers becomes lost in a comparatively early stage in the segmentation, and is not recognisable again until the time when the primordium of the gut is differentiated.

Rossbach's contention that the cells which go to form the

"Hüllmembran" represent the first indication of cell different seems to me to be explaned on the ground that, as is shown i fig. 1, the differentation of the germ layers has already taken for the most part in an earlier stage in the segmentation. figures show also that all cell outlines have been lost, so we conclude that his material was unfavorable for the demonstr of this point in the development.

His figures of the early stages in the development o parthenogenetic eggs (germ balls) within the redia show, how that here at least, the first two segmentation divisions result i cutting off of two smaller cells from a larger one. This proce in itself the separation of the primitive ectoderm from the prim endoderm, and it seems most probable that the same process place in the development of the redia.

The entire absence of cell outlines, or even a separation o cell layers, in most of the embryos, has made it impossible to the cellular history of any of the organs of the larvae with exception of the gut and the investing membrane. In the development the only material which has suggested a possibl cognizable formation of the mesoderm is shown in Fig. 54. Her cells forming the endoderm are surrounded by two concentric masses which are sharply separated from one another. The o most of these surrounding layers is plainly the ectoderm (com Fig. 53), the inner layer may represent the beginning of the r derm; although even if this is admitted, there is not the slig evidence to show from which of the primary germ layers it its origin.

The development of the cercaria.

There are some striking discrepancies in the observatio previous investigators in regard to the order, and, in many inst to the manner, in which the systems of organs become different in the development of the cercaria.

According to Schwarze (1886) the first differentation notic in the development of the cercaria takes place when darkly sta nuclei appear in the interior of the segementing germ ball. T nuclei are most numerous in the central part of the morula, are interpreted as constituting the primordium of the reprodu system. These cells begin to be differentiated before the m has been overgrown by the investing cells.

HASWELL (1903) has pointed out that in other forms (*Echinostomum*) the same appearance of the nuclei is often noted in early stages of segmentation. He states, however, that they are not restricted to the central part of the embryo, and suggests that the characteristic appearance of these nuclei is to be accounted for, not on the supposition that they differ from those of the surrounding cells, but that they represent a certain stage in the usual cycle of cell development.

My own observations on *Diplodiscus* are in perfect accord with those of HASWELL. In any section containing young embryos it is possible to find many nuclei similar to that shown by SCHWARZE in his fig. 1. In a preceding section of this paper it has been pointed out that the densely staining stage of a nucleus is a characteristic step in the cycle of events leading up to its division by mitosis. Such an appearance does not, therefore, afford any certain basis for the assertion that the nuclei noted by SCHWARZE are different from their neighbors.

LOOSS (1892) states that the first of the permanent organs to appear in the young cercariae of *Amphistomum subclavatum* is the water vascular system. He figures (fig. 11 tab. 20) the two main trunks of this system as extending nearly the whole lenghth of the body, which as yet consists only of an undifferentiated mass of "meristem cells".

ROSSBACH (1906), in the passage previously quoted, recognizes the gut as the first of the permanent organs to be laid down. He states that the pharynx makes its appearence at about the same time.

TENNENT (1905) notes first the appearence of a number of large cells which form the primordium of the pharynx.

In the embryos of *Diplodiscus*, developing from parthenogenetic eggs, the primordium of the gut often remains recognizable from the time when the single macromere is left after the cutting off of the two micromeres. In embryos where the limits of the primary germ layers are indistinguishable (Fig. 55), the first organ differentiation to be recognised is the assembling of the endoderm cells to form the primordium of the gut. A definite arrangement of some of the ectoderm cells at the anterior end of the body to form the primordium from which the anterior sucking disc, and the ectodermal portion of the digestive tract, is derived, takes place at practically the same time.

When the embryo has reached the age at which the primor begin to be laid down, the differentiation is very rapid. The cretory bladder and the main trunks of the water vascular sys make their appearence, closely following upon the differentia of the primordium of the gut. The brain and the main branc of the nervous system become recognisable at about the same t The cells which are to form the ventral sucker become marked from the surrounding meristem cells: the large vacuolated along the ventral part of the body (Blasenzellen of Schwarze) m their appearance; and the cuticular gland cells become sha defined. The cells from which the reproductive organs are to developed form a large mass in the central part of the body which there can soon be recognised the primordia of the ovary, testes and the accessory portions of the reproductive system.

The development of the digestive system.

The development of the digestive system, whether the primord of the gut has come over from the primitive endoderm cells recognisable cell mass, or is differentiated from a homogenous cytium is the same. The endoderm cells become arranged in solid rod which is surrounded by the meristem cells. At this s in the development the primordium has much the same appear a has been figured by Rossbach for that of the redia.

Schwarze (1886) and Looss (1892) describe the primordiu the intestine as arising at the anterior end of the embryo, w the mouth opening is to be established, and from the there gro backward to the posterior limits of the caeca. In *Diplodiscus* connection of the gut with the mouth opening is established thr the growning backward of the pharynx and oesophagus, which from the differentation of the primordium at the anterior en the body: from the ectoderm. Schwarze, fig. 4, shows what I be to be the last mentioned structure, which he considered as whole of the digestive system.

The lumen of the gut becomes established by the rearra ment of the cells about a central cavity. For a time the gut sists of a straight hollow tube until it becomes connected with oesophagus by the breaking through of its wall at the anterior The division of the gut into the two caeca and the back growth of the latter takes place after the lumen has been for At first the posterior end of the primordium becomes broadened the lumen extends into the arms forming two flask-shaped cavities. The backward growth takes place from these extremities so that the caeca are developed in their normal positions.

The lumen of the pharynx and oesophagus is established in the same manner as was that of the gut. The oesophagus, which was at first very short, keeps pace with the lengthening of the body. The development of the muscular, posterior, enlargement of the oesophagus takes place after the young cercaria has reached its characteristic form and the parts of the digestive tract have acquired their proportionate lengths.

The anterior sucking disc is formed from some of the cells which were cut off from the remainder of the body when the primordium of the pharynx and oesophagus was marked out. SCHWARZE describes the formation of this sucking disc from cells which become cut off from the surrounding ones by a thin membrane. His figures (fig. 5, 10, 12 and 15) of the anterior sucking disc show it in rather late stages of development only; but on comparing fig. 4 and 5 one is lead to the conclusion that it must have originated from the primordium which in fig. 4 is labeled "*d*" (= primordium of the gut).

The cells concerned in the development of the anterior sucker undergo changes very different from those in the posterior part of the same primordium. In the former cells there appear a large number of fine fibres, which gradually come to fill the greater part of the cell, which is thus transformed into a muscle cell. From their original scattered arrangement they come to lie perpendicularly to the surface of the disc, which in its later development becomes strongly concave. In the dorsal part of the sucker there is developed the dart ("Stachel"). This lies in a thin structureless sheath between the muscle cells. It is shaped like a short arrow with a comparatively broad head.

The dart is made up of an outer hyaline, highly refractive, layer and an inner dark, homogeneous, layer. According to SCHWARZE, with whose observations my own agree, this organ is developed by intracellular cytoplasmic differentiation.

The development of the water vascular system.

As regards the development of the water vascular system in cercariae two views have been set forth which are decidedly opposed to one another. Looss (1892), in describing the development of the cercariae of *Amphistomum subclavatum*, says that the canals arise from "Lückenräume" between the cells of the "primitive parenchym "Zunächst sind Trichter sowohl wie Gefäß nicht aus hohlen Ze zusammengesetzt, wie ich in einer früheren Arbeit anzuneh geneigt war, sondern es sind Lückenräume zwischen den Zell

These spaces run together in a regular manner and by process there are formed two continuous canals running longitudin through the body of the embryo.

As opposed to the view of the "Lacuna Development" of system stand the observations of Bugge (1902), for the cerca and of Rossbach (1906) for the redia. These investigators ag that in the development of both these forms the water vasc canals are formed from primordia which consist of two strands, one on each side of the body. The cytoplasm belongin these cells becomes arranged on one side of the nuclei to for "homogenen einheitlichen Plasmaschlauch". Within this protoplas strand there arises, during the later development, a fine lumen wh forms the water vascular canal.

My observations on the formation of the water vascular sys in *Diplodiscus* are strictly in accord with the last mentioned v In the young embryos (Fig. 56) certain cells on each side of endoderm mass become arranged in longitudinal strands, on outer side of which there is a well defined layer of cytoplasm w in sections appears as a clear thread. The lumen of the canal appears in this clear strand close to the nuclei. As the lume the canal increases in diameter, during the later development, nuclei of some of the cells through which it was formed, com lie in the lumen of the canal. These nuclei apparently degene and are forced out to the exterior.

The terminal portion of this system — the excretory bladder the canal leading from it to the dorsal pore — develops simultaneo with the lateral trunks. The bladder first appears as a circ scribed mass of cells, which are cut off from their neighbors wall of thin cells. Within this mass of cells there soon appea small cavity which rapidly increases in size through the deg ration of the cells within the primordium. The terminal cana formed from a continuation of this same cell mass which ext from the body of the primordium to the dorsal wall of the bod the cercaria.

The flame cells are formed from meristem cells along the co of the canals. At the beginning of the process these cells ma at some distance from the canal in an apparently solid mass of tissue. Before the flame cells have become completely differentiated, however, they are put in communication with the canal through a capillary tube. The lumen of this capillary becomes apparent first near the flame cells, and only later acquires its connection with that of the water vascular trunk.

The reproductive system.

The first indication of the reproductive system is seen after the primordia of the digestive and water vascular systems have been differentiated. A dense mass of cells becomes localized in the median plane of the embryo between the posterior ends of the caeca and the excretory bladder. At first there is no distinction between the parts of this mass which are to give rise to the male and to the female reproductive organs. In the later development of the embryo, this primordium becomes differentiated into a mass of cells which represent the ovary, a mass from which are developed the testes, and a ventrally situated mass which gives rise to the penis with its sheath. These larger masses are connected by strands of cells from which the male and female ducts are developed. In general my observations confirm those of SCHWARZE, except in the matter of the first appearence of the primordium.

The nervous system.

The first differentation of nervous tissue takes place in the region of the future brain. The cytoplasm in a sharply defined area above the primordium of the oesophagus becomes filled with very fine fibres among which there may be distinguished a few scattered nuclei. These nuclei differ very markedly in appearance from those of the surrounding meristem cells. The limits of the nervous tissue becomes marked out by the arrangement of the parenchyma nuclei about its periphery to form the rudimentary sheath. The two bilateral masses of nervous tissue — the lobes of the brain — become connected together by a strand which arises through the fusion of two outgrowths, one from either mass. The main branches of the nervous system arise as anterior and posterior outgrowths from the brain. In none of my material have I been able to recognise the pigmented eye spots which have been described for *Amphistomum subclavatum* by LOOSS (1892).

The cystogenous glands.

The cystogenous glands arise at a comparatively late stage in the development of the embryo, through a transformation of the meristem cells in an area along either side of the body. These cells increase greatly in size and their contents become highly granular, while their cytoplasm becomes restricted to the interstices between the granules of the secretion.

The cuticula.

The origin and nature of the cuticular covering of trematodes has been one of the most discussed questions concerning their histology. The opinion which has received the most support among modern investigators, is that the covering is a true cuticle, i. e. the product of an epidermis. The epidermis is believed to be represented by deeplying glandular cells, whose ducts traverse the muscular layers and the basement membrane to pour their secretion on the outer surface of the latter. According to some observers, the original covering of the embryo (the investing membrane), goes to make up the basement membrane of the adult worm.

In sections through young embryos before the cuticula has been formed, there are found near the outside of the body a considerabl number of large cells with deeply staining contents. In favorabl sections the mouths of these cells can be traced through th outer tissues to the basement membrane. After the cuticula ha been formed the above mentioned cells are very different in appearence. They no longer stain deeply with haematoxylin, while th newly formed cuticula has the same staining reaction that wa shown by the contents of these cells before the cuticula had been formed.

The evidence gathered in the study of the formation of th cuticle in this species confirms the view as to the true cuticula nature of the outer body covering as first set forth by BLOCHMAN and KOWALEVSKI.

The body parenchyma.

The body parenchyma cells arise by a direct transformation o the meristem cells of the embryo. The cytoplasm of these cells which in the young cercaria forms a continuous mass, in which th nuclei are imbedded, later becomes broken up into branches whic interlace and anastomose. Between the branches many small cavities are left, which in the living worm are filled with fluids.

The body musculature.

The only observations I have been able to make on the development of the muscles are as follows: in young cercariae, when the other organs were clearly marked out, it was noticeable that some of the cells lying just beneath the investing membrane became differentiated from the surrounding cells by a characteristic appearence. The nuclei were arranged on the inner side of a mass of cytoplasm in which there appeared fibrillae, which were most noticeable in longitudinal sections. In none of my material could I trace the later stages in the transformation, nor, indeed, in the fully developed cercariae did I succeed in demonstrating the muscular structures which have been described by Bettendorf (1897).

3. The development of the tail of the cercaria.

In early stages of the development of the cercaria, when it becomes elongated and the anterior end is recognisable through the formation of the primordium of the anterior sucking disc, the posterior end becomes considerably narrowed. This narrow portion continues to elongate as the embryo increases in size, until it eventually becomes about three fourths the length of the body. When first clearly distinguishable, the tail consists entirely of a mass of meristem cells. In its later development, there is a special differentation in three regions. Some of the cells near the surface become differentiated into epidermal cells and secrete a cuticula similar to that which covers the body of the cercaria. Just internal to the basement membrane (investing membrane) there is developed a thin layer of longitudinal muscles. At the centre of the tail there is a central axis of muscle fibres, which is at many points connected with the muscular layer beneath the basement membrane. The bulk of the tail is made up of a mass of parenchyma cells which resemble the large vacuolated cells (Blasenzellen) which are developed in the ventral part of the body of the embryo. The tail is at first a direct continuation of the body of the embryo. In later stages of development it becomes separated from the body by a cutinized wall, except along the edges where it is connected by a number of muscular fibres, the so-called "connecting strands".

Part IV.

General Considerations.

1. The nuclear division in *Diplodiscus.*

The character of the nuclear division in this worm differ
many respects from that which commonly occurs among the meta
and in just these points of difference it resembles the type of
sion commonly found among the protozoa.

In the fact that the entire chromatin content of the nucle
gathered into a caryosome, the resting nucleus of *Diplodiscu*
sembles that of many of the protozoa, especially the flagellata
sporozoa.

The persistent intranuclear position of the centrosomes, i
segmentation divisions as well as in the maturation divisions, d
guishes the mitosis in this species from almost all instances a
metazoa. Among the protozoa, on the other hand, the mito
Euglypha alveolata, as described and figured by SCHEWIAKOFF (1
presents a series of nuclear phenomena which, except for the
tribution of the chromatin in the resting nucleus and the form
of the spireme, almost exactly duplicates that found in a seg
tation mitosis in *Diplodiscus*. The persistence of the nuclear
brane throughout the cell cycle, while a rather common occu
among the protozoa, is unknown in the normal mitosis among me
and this fact makes a sharp distinction between the type fou
Diplodiscus and that found in other metazoa.

A comparison of the figures given by many of the worke
the development of the eggs from the parthenogenetic genera
of the Malacocotylea makes it seem probable that intranuclea
tosis is of common occurence among the members of this fa
Most of the investigators above mentioned have made no acc
study of the nuclear phenomena of division, but in many of
figures chromosomes are shown within a sharply defined nu
membrane so constantly that it seems justifiable to conclude
the mitosis in these species in similar to that occuring in
discus.

The evidence from figures of many celled embryos (germ
is especially convincing, for in such cases the nuclei lie in a
of cytoplasm in which there are no cell walls so that there ca

no possibility of confusing the outline of the nucleus with the boundaries of a cell.

In the development of the fertilized egg, according to the observations of all modern investigators, the mitosis is similar to that in other metazoa. In the later stages of the development of the sporocysts of *Diplodiscus* the mitoses were of the same form as those found throughout the later stages in the life history. In the earliest cleavages of the fertilized egg it seems probable that the nuclear membrane must be ruptured to allow the fusion of the male and female elements. In view of this last mentioned fact, it would seem probable that within the life cycle of a single worm there may be both the intranuclear and extranuclear types of division: although in any one species this has not been determined with certainty.

In the fertilized eggs of the Heterocotylea, and in those of *Fasciola hepatica*, at least, among the Malacocotylea (Schubmann, 1905), the centrosomes lie outside the nucleus. In most figures of the development of the rediae and cercariae no mention is made of these structures, but the apparently intranuclear type of the mitosis would imply the intranuclear position of these bodies.

The behavior of the chromosomes in the anaphase and in the telophase of mitosis necessarily differs in *Diplodiscus* from what usually takes place in metazoan nuclei. In this worm the chromosomes never form chromosomal vesicles, but fuse together to form the dense caryosome, which, even with the best obtainable differentiation, appears perfectly homogenous, from the late anaphase stage to the beginning of its increase in size in the early prophase. In this feature of the mitosis the resemblance to the mitosis in protozoa s again very marked.

2. The Life History of the Malacocotylea.

In the light of the results recorded in this work the life history of the Malacocotylea is shown to be one in which usually two parthenogenetic generations are interpolated between any two generations coming from fertilized eggs. The old view that in the life history of these forms there was a true alternation of generations — Metagenesis — as well as that of Leuckart, Balfour and Looss, who saw in this process a metamorphosis extending over several generations, must be given up. Claus' interpretation of the life cycle as heterogeny with paedogenesis seems to have the most facts in its support.

Looss has pointed out that the three forms, sporocyst,
and cercaria are built upon the same fundamental plan and
the essential differences between them is in the increasing compl
which is shown in the structure of the three members of the s
in the order named. This is most clearly shown in the develop
of the digestive system, the water vascular system, and in the
vous system; while the structure of the reproductive system
the cercaria sharply over against the parthenogenetic forms.

To draw a comparison from the development of the cercari
is as though the development of the body of the sporocyst
stopped when the primordium of the gut had first appeared an
water vascular system had developed to a stage when only
main trunks with a single flame cell attached to each had
differentiated; while the redia represents a stage in which the
lopment has gone on until the gut consists of the unpaired primord
the anterior sucker and the pharynx and oesophagus had been
loped, and the water vascular system had become branched,
the body has become elongate in outline.

Except for the presence of mature sexual elements, the
parthenogenetic generations represent larval forms in all
structure.

The power of reproduction which has been developed in
larval forms may be interpreted as a special provision for a
crease of individuals which is necessitated in order to compen
for the fact that there is only a very slight probability that
cercaria will meet with the conditions necessary for its fu
development. If only a single individual came from each ferti
egg, there would be small chance that, with its complicated
history with the consequent number of critical stages, enough emb
would be able to transform to the adult condition to maintain
life of the species.

Summary.

1. *Diplodiscus temporatus* Stafford differs in many charac
particularly in the nurse generations and the cercaria, from
European species *Amphistomum subclavatum* with which it has
generally confounded.

2. For the formation of a complete cyst it is essential that
cercaria become attached to some substratum by means of the

tral sucker; otherwise only an open tube of the cyst forming material will result.

3. The cercariae are positively heliotropic, and when set free from the snail in a normal manner, always swim to, or near to, the surface before encystment takes place.

4. The encysted worms are unable to withstand dessication for more than one hour.

5. Tadpoles, or other animals which obtain their food by browsing along the banks of streams, may take up the encysted cercariae with their food. In large tadpoles the worms become transformed into sexually mature worms. In other animals, and in some cases at least in small tadpoles, the worms become again encysted. If such intermediate hosts are eaten by adult amphibians the worms would be able to go on with their development again.

6. The eggs (germ cells) in the sporocyst may arise either from cells which have come over from an early developmental stage free in the body cavity of the sporocyst, or they may as is the case throughout the later life of the sporocyst, arise from nuclei in the body wall which become segregated at one end of the body cavity to form a rudimentary ovary.

7. The mitosis in this form is intranuclear. All of the chromatin in the nucleus is gathered into a single (rarely there are two) caryosome. This body is surrounded by a densely staining mass of cytoplasm. The centrosomes in the resting stages of the nucleus lie either in the body of the caryosome, or so closely applied to it that their identity cannot be made out. The centrosomes become apparent in a comparatively late prophase.

In the prophase, the caryosome increases in size at the expense of the nucleoplasm until the whole of the nuclear contents are gathered into a single, lightly staining, mass which is connected with the nuclear membrane by several strands. Within this, at first homogenous mass, there appear chromatin granules which later increase in size and finally fuse to form the spireme thread, which soon segments to form the chromosomes.

The spindle fibres are very prominent, each one being apparently made up of several (four) smaller fibres.

Up to the metaphase of division the nuclear membrane retains its nearly spherical shape. The elongation begins in the anaphase and the constriction follows immediately.

In the anaphase the daughter chromosomes are drawn up into a compact mass, in which all outlines of the separate chromoso are soon lost.

The constriction of the nuclear membrane continues until daughter nuclei are separated as though by amitosis.

8. The formation of the spireme for the maturation divi takes place without the concentration of the whole of the nuc contents into an enlarged caryosome. Instead, the caryosome comes directly converted into the spireme thread without the in vention of a stage in which the chromatin exists is small granu

Usually the maturation spindle is excentric in the egg; it located near that end of the nucleus which lies nearest to cell wall.

In the formation of the polar body a portion of the nucl membrane containing one set of the daughter chromosomes from maturation division becomes extruded from the cell so that the p body contains no cytoplasm, but consists of a portion of the nucl surrounded by a double membrane.

9. The mitosis in this form resembles closely that occurin the protozoa.

10. The two micromeres, which represent the ectoderm, given off from the single macromere (mes-entoderm), in the first divisions. The gastrulation is epibolic. Two of the ectoderm c in the nine cell stage, are set apart from the rest to form the vesting membrane. The embryo soon becomes a syncytium, but separation between the ectoderm and endoderm usually rem distinct for some time after the cell walls are lost. Someti another layer of cells (nuclei and cytoplasm) may be distinguis between the ectoderm and endoderm.

11. The primordium of the gut often retains its identity f the stage when it exists as a single cell after the separation of two primary micromeres. The primordium of the anterior suc disc, pharynx and oesophagus becomes marked out at the ante end of the embryo. The water vascular system arises as two lat intracellular tubes, one on either side of the body. The flame arise in connection with some of the meristem nuclei and later quire their connection with the lateral trunks.

The reproductive organs arise first as a single mass of c which later becomes separated into recognisable primordia the testes, the ovary and the accessory parts of the reproduc system.

12. The life cycle of the Malacocotylea may be characterised as Heterogeny with Paedogenesis.

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Erklärung der Abbildungen.

Acet acetabulum
as anterior sucker
br brain
caec caecum
cent centrosome
cf connecting filaments
cg cystogenous glands
cgc cystogenous gland cells
cgr chromatin granules
chr chromosomes
cm cyst material
cn commissures
cu cuticular covering of the body
cyt cytoplasm
e egg
ect ectoderm
end endoderm
exb excretory bladder
exc excretory canal
exp excretory pore
g gut
gb germ ball
gp genital pore
invc investing cells
izf interzonal filaments
k caryosome
ltn lateral nerve trunks
mac macromere
mc circular muscles
mic micromere
ml longitudinal muscles
mt transverse muscles
mth mouth
nm nuclear membrane
np nucleoplasm
nu nucleus
oes oesophagus
ov ovary
ovw wall of ovary
par parenchyma
pb polar body
php pharyngeal pockets
pla posterior locomotor appendages
prg primordium of the gut
prph primordium of the pharynx
prov primordium of ovary
prt primordium of testes
prut primordium of uterus
s spireme
sg shell glands
spf spindle fibres
st spireme thread
sts „suspensory strands"
t posterior testis
t. l anterior testis
tl tail
ut uterus
vd vas deferens
vit vitellaria
vs ventral sucker
wvt main trunks of water vascular syst
y. cerc young cercariae, in body cavity of redia

Unless otherwise stated, all figures are drawn with camera lucida at the magnification obtained with the ZEISS immersion objective 1,5 mm and compensating ocular 12.

Plate 30.

Fig. 1. Adult worm somewhat contracted, drawn from the ventral side as a transparent object. ZEISS Obj. 16 mm and Comp. ocular 4.

Fig. 2. Same specimen drawn from the dorsal side. Same magnification.

Fig. 3. Sporocyst from liver of *Goniobasis*. ZEISS Obj. 8 mm and Comp. Ocular 4.

Fig. 4. Redia from liver of *Goniobasis*. Same magnification as last figure.

Fig. 5. Encysted cercaria. ZEISS 8 mm and Comp. ocular 4.

Fig. 6. Mature cercaria drawn as a transparent object from the dorsal side. The cystogenous glands are omitted on the right side of the figure. ZEISS Obj. 8 mm and Comp. Oc. 4.

Plate 31.

Fig. 7. Slightly diagonal longitudinal section through a mature cercaria before encystment, to show the external opening of the water vascular system. ZEISS Obj. 4 mm and Comp. Oc. 4.

Fig. 8. Transverse section through the body of a nearly mature cercaria which had given off the contents of the cystogenous glands while still within the body cavity of the mother redia.

Fig. 9. Section through a very young sporocyst.

Fig. 10. Section through an older sporocyst before the formation of the ovary (Keimlager). ZEISS Obj. 1,5 mm and Comp. ocular 6. $^1/_3 : 1$.

Fig. 11. Section through a sporocyst at the time of the localization of the germinal nuclei to form the ovary. Magnification as in the last figure.

Fig. 12. Section through sporocyst in slightly later stage than in the previous figure. Magnification as in last figure.

Fig. 13. Section through ovary of fully developed sporocyst. Magnification as in last figure.

Plate 32.

Fig. 14. Nucleus of segmentation cell in the resting condition.

Fig. 15. Nucleus of segmentation cell in early stage of spireme formation.

Fig. 16. Nucleus of segmentation cell in later prophase.

Fig. 17. Later prophase, first appearance of the chromatin granules.

Fig. 18. Segmentation nucleus in which the redistribution of the nucleoplasm has begun, and in which the chromatin granules have increased in size and are fusing to form the spireme thread.

Fig. 19. Nucleus in which the spireme thread has been formed and already extends beyond the circular mass of nucleoplasm.

Fig. 20, 21, 22. Three successive stages in the growth of the spireme and the redistribution of the nucleoplasm.

Fig. 23. Nucleus containing fully formed spireme.

Fig. 24. Nucleus in which the segmentation of the spireme to form the chromosomes has been nearly completed before the redistribution of the nucleoplasm has taken place.

Fig. 25. Nucleus in which all of the chromosomes are distinct, but still attached to one another by linin filaments.

Fig. 26. Polar view of a nucleus in the metaphase.

Fig. 27. Side view of a nucleus in which the chromosomes are being arranged to form the equatorial plate.

Fig. 28. Side view of a nucleus in the metaphase.

Fig. 29. Side view of a nucleus in rather later anaphase to show the beginning of the constriction of the nuclear membrane.

Fig. 30. Cell in which the division of the nucleus is nearly completed.

Fig. 31. Egg showing the characteristic shape of those which come from the ovary in the sporocyst.

Fig. 32. Egg showing fully formed spireme for the maturation division.

Fig. 33. Egg in a late prophase of the maturation mitosis, the spireme thread has segmented to form the chromosomes, several of which are distinctly shown as separate.

Fig. 34. Side view of an egg at the metaphase of the maturation division.

Fig. 35. Section, in plane of the equatorial plate, through an egg in the metaphase of the maturation division.

Fig. 36. Side view of an egg (which underwent its maturation while free in the body cavity of the sporocyst), in an early anaphase of the maturation division.

Fig. 37. Side view of an egg in late anaphase. (Egg matured free in body cavity of the sporocyst.)

Fig. 38. Egg in early telophase of maturation division.

Fig. 39. Late telophase of the maturation division.

Fig. 40. Egg with attached polar body in which the chromosomes are still distinct from one another after those in the egg have fused to form the caryosome.

Fig. 41. Egg with two polar bodies (?) attached.

Plate 33.

Fig. 42. Egg in resting stage before the first segmentation division.

Fig. 43. Section, oblique to plane of equatorial plate, through egg at metaphase of the first segmentation division.

Fig. 44. Side view of an egg at the metaphase of the first segmentation division.

Fig. 45. Section through equatorial plate of egg in first cleavage.

Fig. 46. Two cell stage, following first cleavage.

Fig. 47. Three cell stage, result of second cleavage.

Fig. 48. A four cell stage, resulting from irregular segmentation of the micromeres.

Fig. 49. Five cell stage: the usual result of the third cleavage.

Fig. 50. Section through an embryo showing the formation of the investing membrane.

Fig. 51. Slightly older stage, showing the characteristic degenerative changes in the nuclei of the investing cells.

Fig. 52. Section through an embryo in which all of the cells, with the exception of the endoderm cells, have lost their outlines.

Fig. 53. Section through an embryo in a later stage of development; the endoderm is clearly marked off from the ectoderm.

Fig. 54. Section through an embryo in which there is a well defined layer of cells between the ectoderm and the endoderm.

Fig. 55. Section through an embryo in which there is no distinction between the cell layers, except for the greater size of the endoderm nuclei.

Fig. 56. Longitudinal section through an embryo showing the primordia of the gut, of the anterior sucking disc and pharynx, and of the water vascular system.

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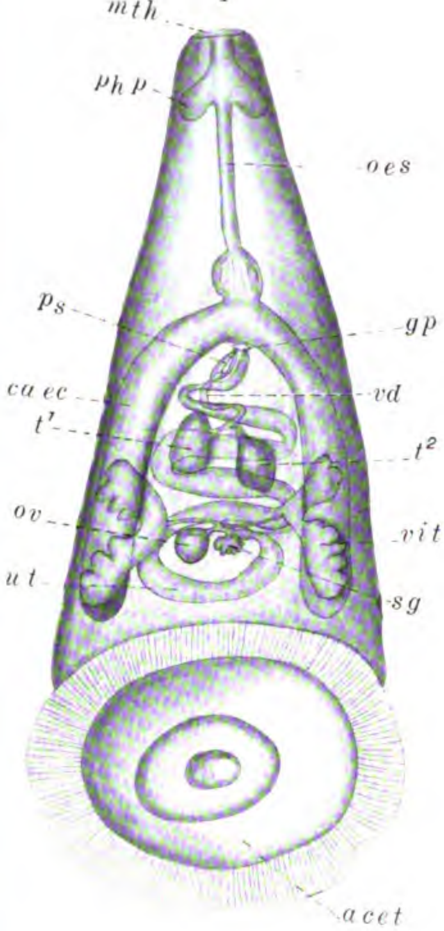
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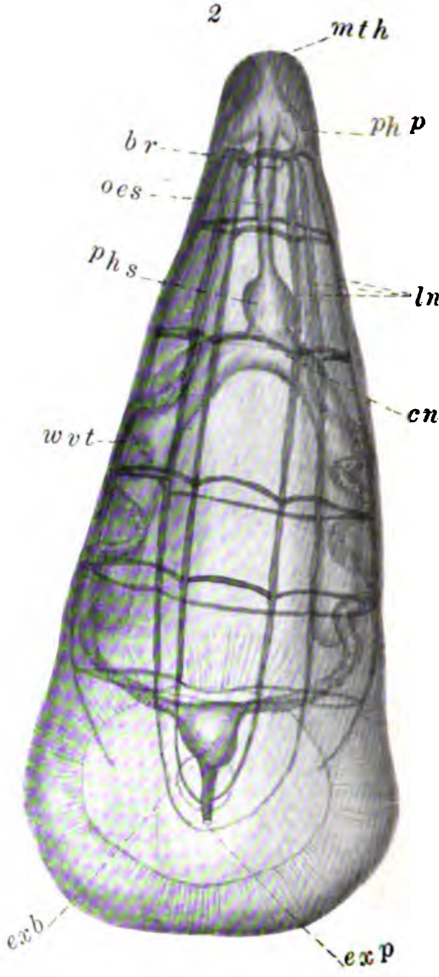
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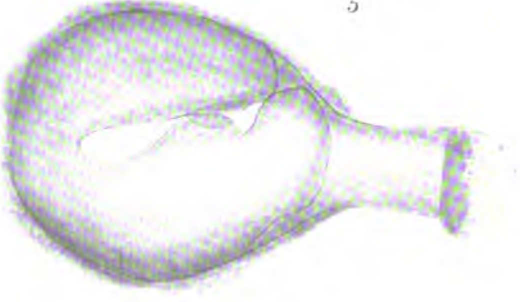

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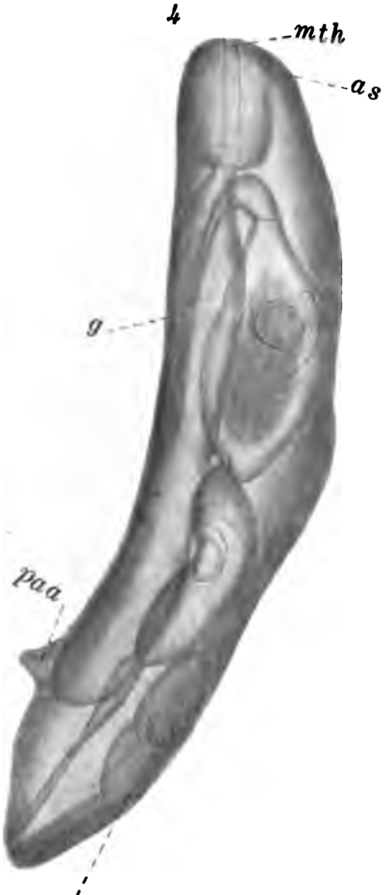

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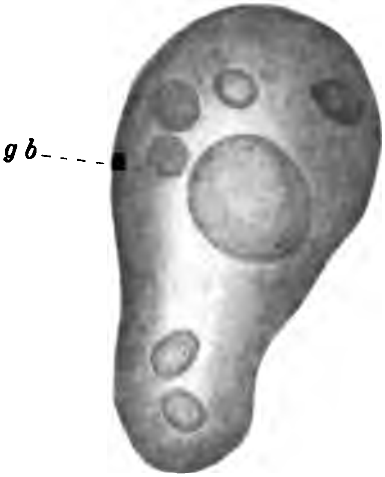

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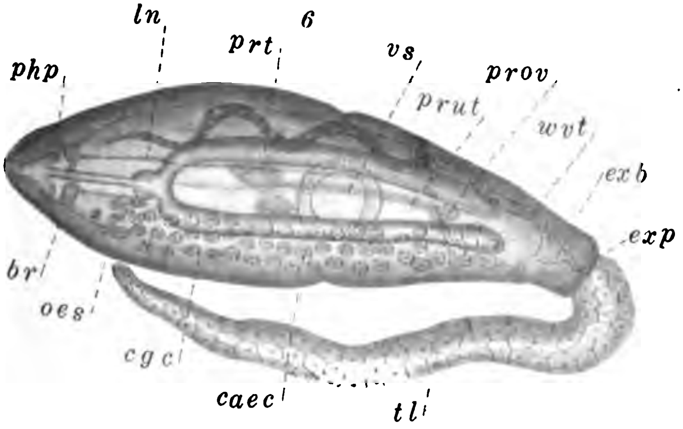

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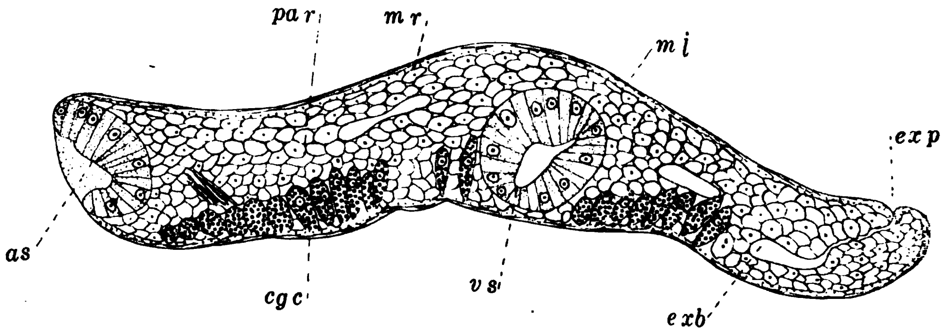

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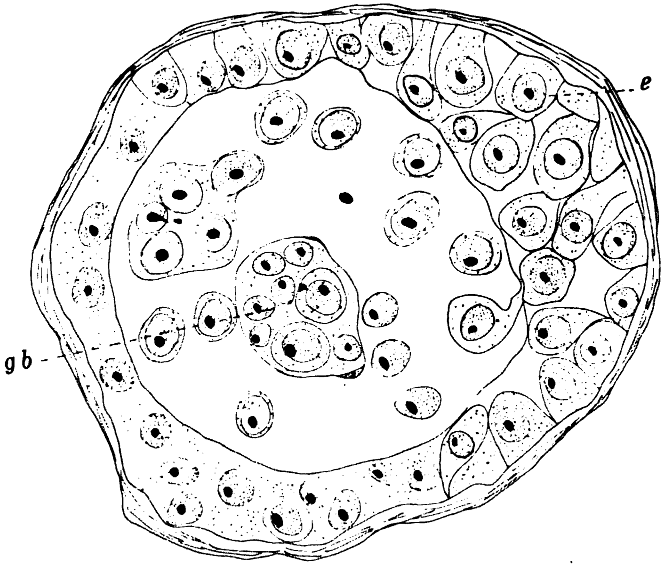

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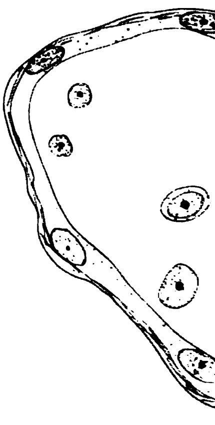

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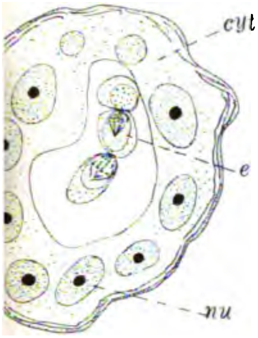

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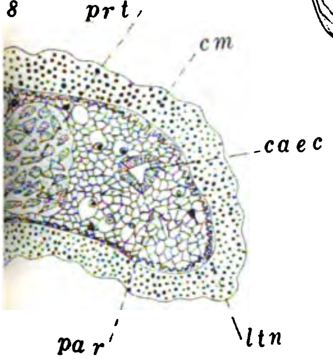

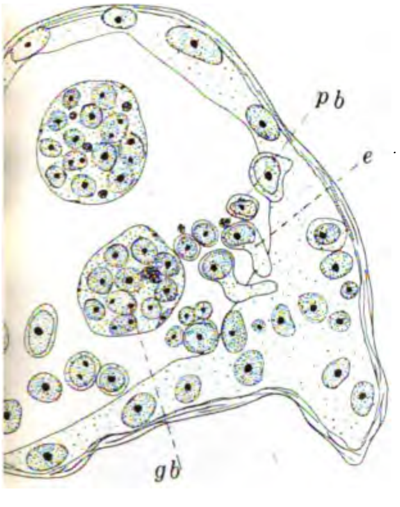

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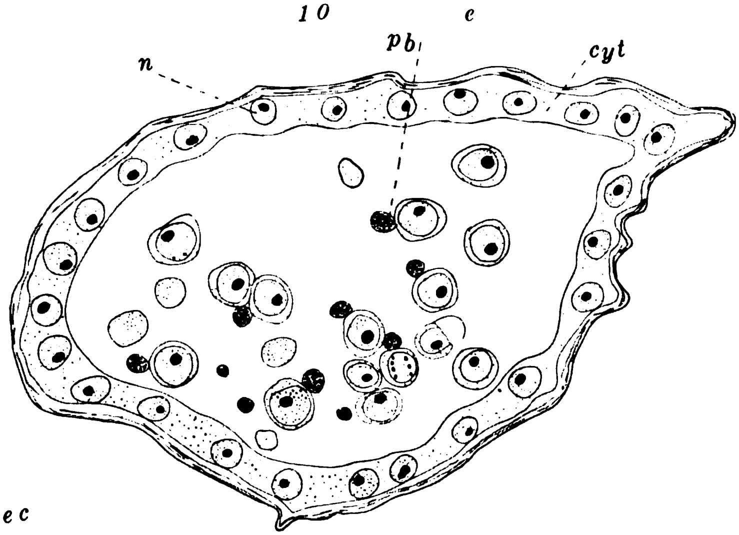

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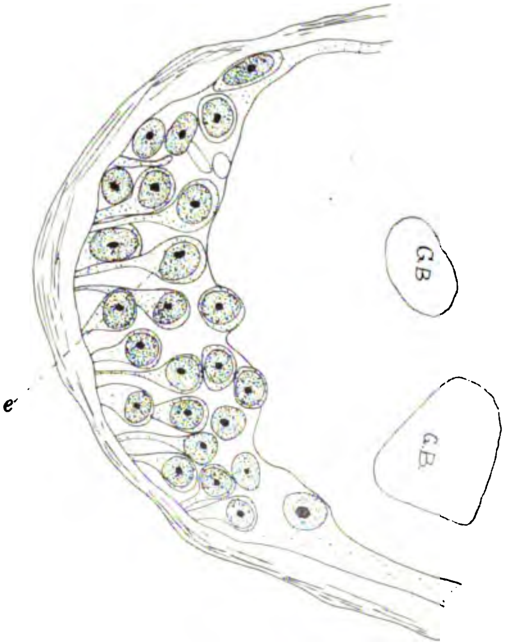

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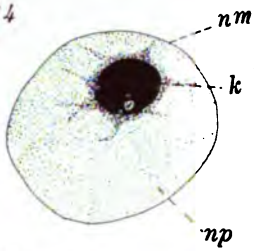

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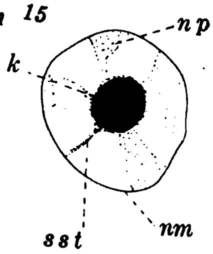

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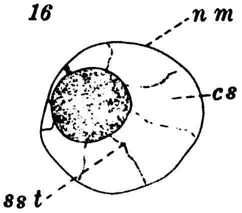

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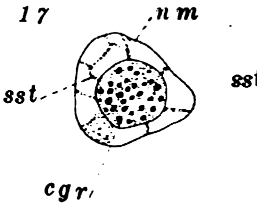

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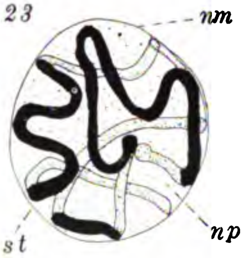

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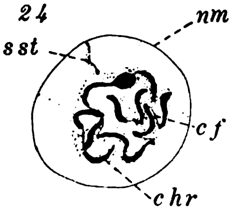

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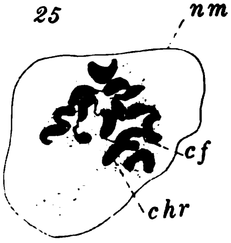

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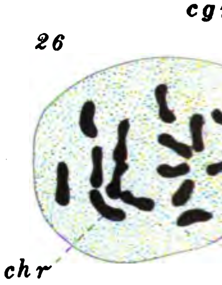

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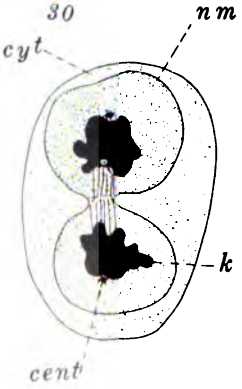

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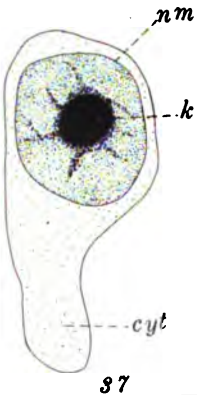

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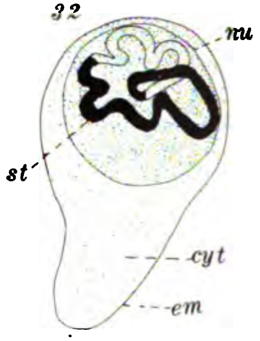

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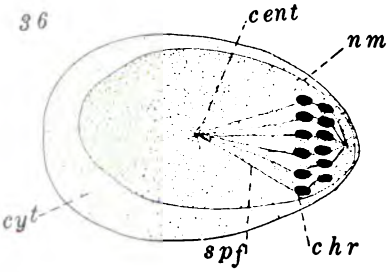

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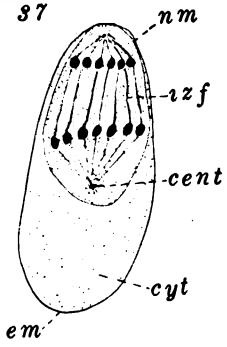

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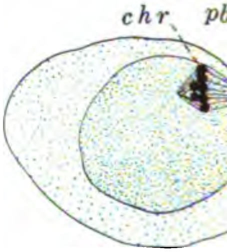

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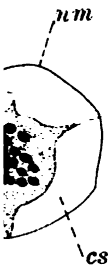

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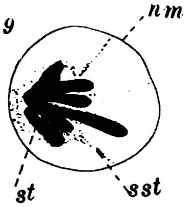

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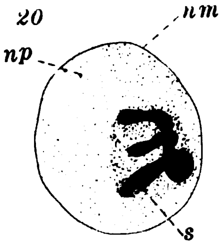

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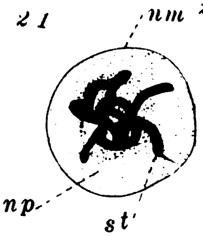

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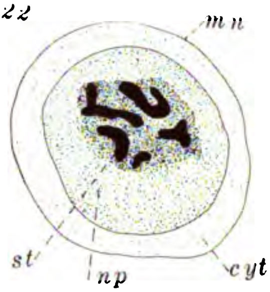

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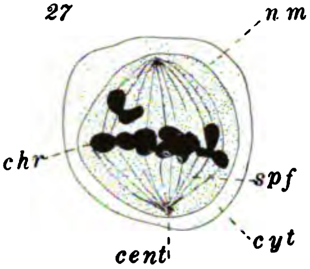

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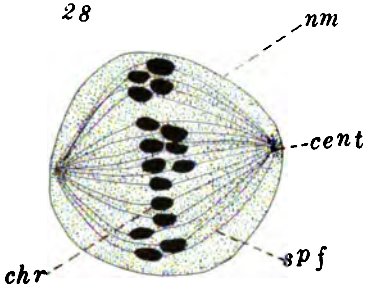

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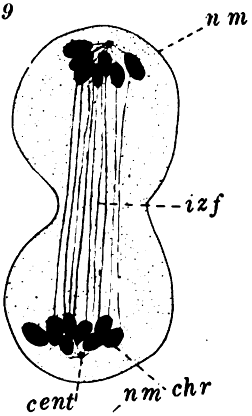

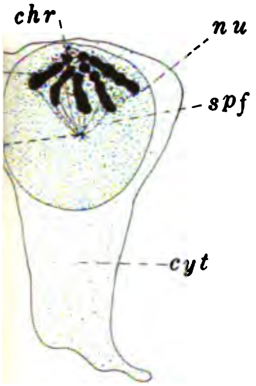

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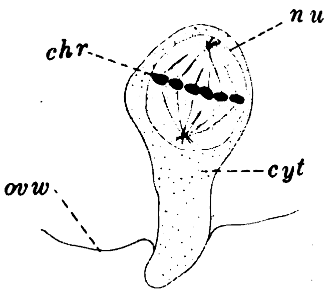

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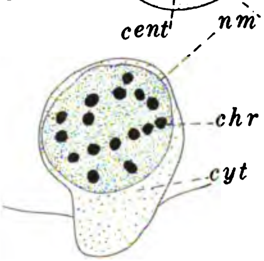

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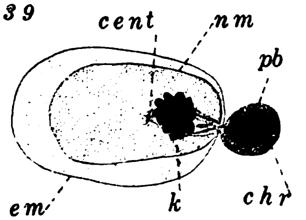

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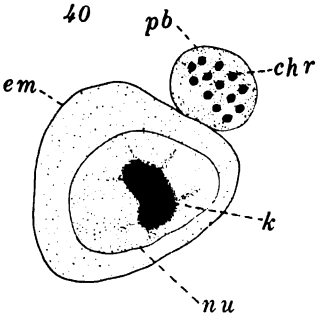

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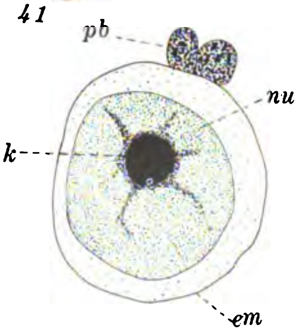

Fischer in Jena.

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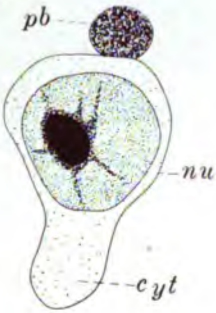

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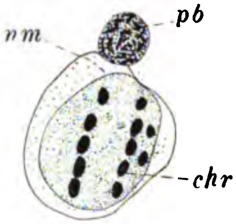

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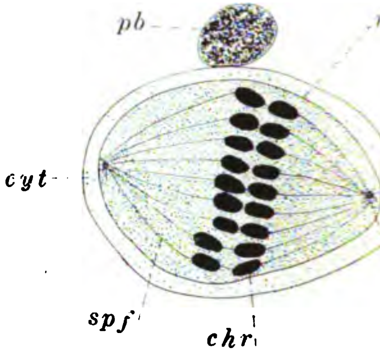

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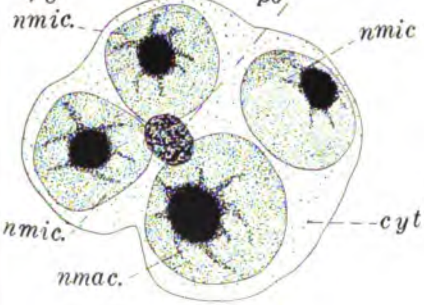

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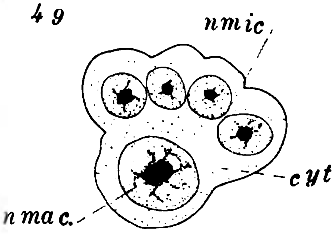

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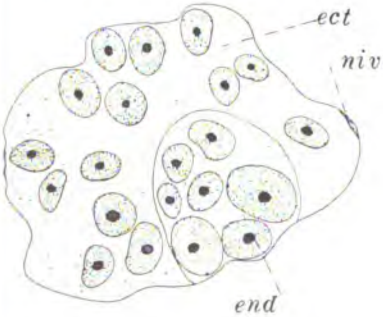

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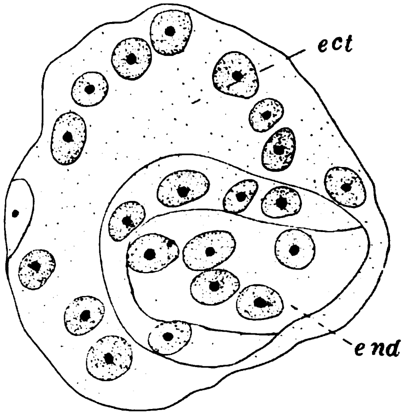

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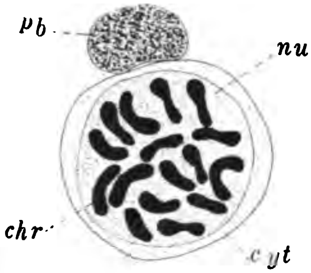

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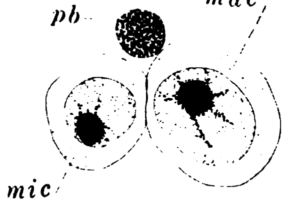

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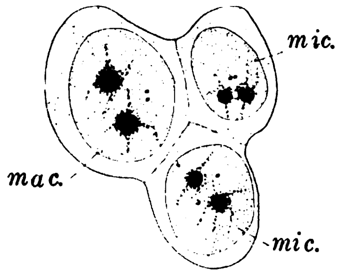

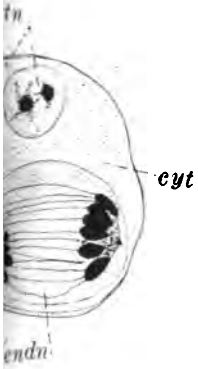

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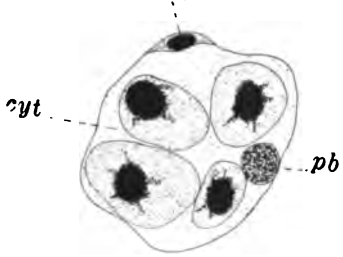

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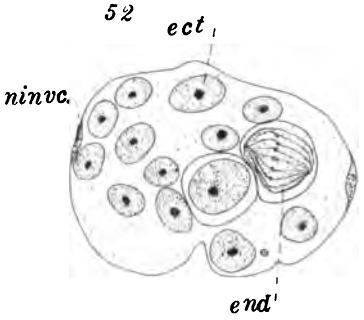

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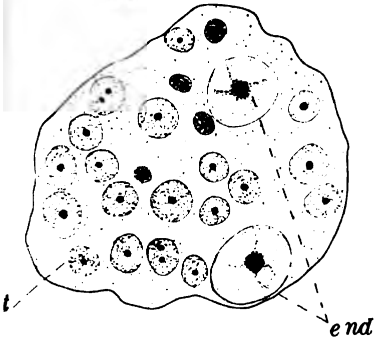

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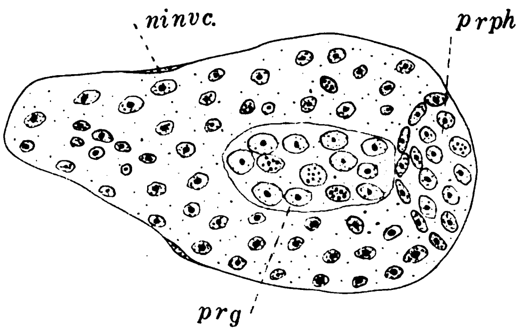

Fischer in Jena.

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1928. Riverente omaggio dell'autore

Clinica Dermosifilopatica della R. Università di Pavia
Diretta dal Prof. Umberto Mantegazza

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DOTT. ROBERTO CASAZZA
ASSISTENTE

SUI DERMATOENDOZOI,

a proposito di un nuovo parassita.

(Creeping disease e Pseudo-scabbia da *Dermolecanium migrans*)

Dal *Bollettino della Società Medico-Chirurgica di Pavia*
Anno XLII dalla fondazione della Società
1928 (a. VI) - Fascicolo 3

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(Creeping disease e Pseudo-scabbia da " Dermolecanium migrans ,,) (*).

(Comunicazione fatta nella Seduta del 27 aprile 1928)

INTRODUZIONE

Molte e varie sono le specie appartenenti al regno animale — ed assai ampiamente scaglionate nella scala zoologica — le quali si dimostrano parassite della pelle umana. Lasciando da parte i Protozoi (gli esseri unicellulari) che hanno tutta una patologia propria ed importantissima, è pur sempre assai grande il numero dei Metazoi (pluricellulari) che parassitano la cute ed il tessuto sottocutaneo dell'uomo: ai fini particolari del presente lavoro è necessaria anzitutto una divisione di questi Metazoi in due grandi gruppi, comprendenti da una parte quelli che vivono alla superficie del tegumento umano, dall'altra quelli che vivono — esercitando il loro paras-

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(*) Comunicazione precedentemente fatta alla XXIV riunione della Società Italiana di Dermatologia e Venereologia. Roma, 3 aprile 1928 - Anno VI.

sitismo — nello spessore della cute o del sottocutaneo, che essi ledono nell'integrità anatomica. Per il primo gruppo Darier usa la denominazione di « epizoari », di « dermatozoari » per i secondi — e similmente Riecke — mentre Dubreuilh (in *La Pratique Dermatologique*, 1900), usava indistintamente la dizione « dermatozoari ». Per maggiore chiarezza ci serviremo della classifica di ***dermatoepizoi***, per il gruppo dei parassiti di superficie, e di ***dermatoendozoi*** per il secondo gruppo. Il primo, che è anche il più numeroso di specie, non è interessante per la nostra esposizione: verso il secondo invece ci rivolgeremo massimamente, con lo scopo di portarvi un modesto contributo di osservazioni.

Le nostre conoscenze sui dermatoendozoi sono in parte di data assai antica, in parte assai recenti; le specie parassite più anticamente note appartengono agli Artropodi, mentre gli Elminti, che pure li precedono nella scala zoologica, vennero osservati solo in seguito. Perciò anche seguiremo quest'ordine nella trattazione.

Gli Artropodi dermatoendozoi.

Gli **Artropodi Tracheati** (¹) si dividono in tre classi: Aracnidi, Miriapodi e Insetti, delle quali la seconda (Miriapodi) non ha interesse dal nostro punto di vista.

Nella prima classe — ***Aracnidi*** — l'ordine **Acari** fra le molte ci presenta due famiglie, fam. ***Sarcoptidi*** e fam. ***Demodecidi***, che racchiudono in sè rispettivamente il *Sarcoptes scabiei var. hominis* (un dermatoendozoo fra i

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(¹) Seguo quasi assolutamente, per tutto quello che è classificazione, l'ultima edizione (1927) dei *Précis de Parasitologie* del BRUMPT (Masson e C., Parigi) pur non ignorando che questa classificazione non corrisponde in tutto a quella adottata dalla maggior parte dei ditterologi.

più anticamente noti) e il *Demodex folliculorum var. hominis*. Dico senz' altro che il *D. folliculorum var.. hom.* è un dermatoendozoo un po' speciale, forse da considerarsi spurio, in quanto vive entro la pelle, è vero, ma in una cavità naturale (follicolo sebaceo), non in una cavità scavata « ad hoc » a spese dell' integrità dei tessuti cutanei. Ma, ritornando alla famiglia dei *Sarcoptidi*, in essa oltre al *S. scabiei var. hom.* e al *S. scab. var. crustosae*, che sono gli agenti causali rispettivamente della scabbia volgare e della scabbia norvegese, *e che sono fra i più tipici dermatoendozoi* (¹), noi troviamo altre varietà, d' origine animale, che possono passare sull' uomo e dare dei quadri clinici a tipo scabbia, ma nei quali — oltre le notevoli differenze cliniche — *quasi sempre si nota l' assenza di cunicoli*. Il parassita vive ed opera così in superficie; si comporta quindi quasi esclusivamente da dermatoepizoo. Queste specie sono: il *Sarcoptes scabiei var. equi* (scabbia equina, con aspetto di pityriasis rubra (Darier)), *S. sc. var. ovis, var. caprae, var. aucheniae, var. cameli, var. suis, var. canis, var. vulpis, var. leonis*, il *Sarcoptes minor var. cati* (scabbia sarcoptica del gatto) e il *Chorioptes symbiotes var. bovis*. Quest' ultimo fu trovato da Moniez (citato da Brumpt) nei follicoli sebacei del naso, presentando così una localizzazione tutt' affatto simile a quella che ha richiamato la nostra attenzione a proposito del Demodecide dei follicoli. E ancora è molto interessante quanto dice Brumpt, che cioè in Africa, negli individui colpiti da scabbia comune, è spesso impossibile trovare cunicoli. A quale causa

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(¹) Nella scabbia norvegese si possono avere cavità contenenti acari le quali si approfondano fino nello strato spinoso. Riguardo poi al parassita di questa forma morbosa, è tuttora " sub judice „ la questione se esso sia identico o no al *Sarcoptes scabiei var. hominis*. Ho mantenuta la divisione fra le due varietà in omaggio a quanto fa il Brumpt senza voler affatto pronunciarmi sulla questione.

attribuire questo epizooarismo? Forse a variazioni della pelle in quegli individui, come vediamo — in patologia cutanea — negli psoriasici che Samberger (¹) non ha mai visti affetti da scabbia? O a variazioni del parassita o dell'ambiente? A noi basta l'aver ricordato il fatto.

Abbiamo dunque già tenuto parola delle due prime classi degli Artropodi Tracheati, le classi cioè degli Aracnidi (ottopodi) e dei Miriapodi. Veniamo ora alla terza, quella degli ***Insetti*** (esapodi), fra tutte la più ricca di specie a noi interessanti. Prima di venire alla trattazione di essa, occorre fare un importante rilievo: mentre nelle forme di cui sopra trattammo e dovute ad Acari — siano essi Demodecidi o Sarcoptidi — chi determina la lesione è sempre la forma adulta, invece, nelle forme di dermatoendozoarismo dovute agli Insetti, chi agisce è quasi sempre (vedremo l'eccezione) il parassita allo stato di larva.

Veniamo dunque agli Insetti, e più specialmente agli Insetti Pterigoti, che soli ci interessano. Fra i vari ordini in cui essi si dividono, ci soffermeremo dapprima su quello dei ***Ditteri,*** e più precisamente tratteremo dei ***Ditteri Ciclorafi Schizofori,*** che raccolgono essi soli molte più specie dermatoendozoarie che non gli altri ordini che li precedono e li seguono nella classificazione degli Insetti.

I Ditteri C. S. provocano nella patologia umana, per mezzo di varie specie, numerosi ed importanti quadri clinici, noti sotto il nome generico di Myiasi (²), le quali

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(¹) Citato da DARIER: " Précis de Dermatologie ",, ed. 1923, p. 114.

(²) Degni di ricordo sono gli importanti contributi italiani alla questione, che sono raccolti da V. RAMAZZOTTI nel suo lavoro: " Myasis dermatosa oestrosa da Larva Migrans " (primo caso, se non erro, di vera Creeping disease in Italia), pubblicato nel *Bollettino delle Specialità Medico-Chirurgiche di Milano*, 1927, fasc. II. Fra i più recenti lavori ricordo anche: C. VENERONI: " Myiasi nella Somalia Italiana " *Rif. med.*, 1926, vol. 42, pag. 416.

possono interessare altri e più organi e sistemi all' infuori della pelle. Brumpt infatti ne fa 3 gruppi in base alle localizzazioni:

1° **Myiasi cutanee;**
2° **Myiasi cavitarie;**
3° **Myiasi intestinali.**

Ora, a chi si occupa di Dermatologia interessa evidentemente in massimo grado il primo gruppo, pur tuttavia anche gli altri possono offrire aspetti clinici a lui interessanti. Infatti, nelle **Myiasi cavitarie** (2° gruppo), oltre alle varietà chiamate *Nasomyiasi*, *Otomyiasi* — (appunto per le localizzazioni) — *Oculomyiasi*, *Cistomyiasi*, *M. simultanea delle cavità naturali*, noi vediamo descritta una *Myiasi generalizzata* (eccezionale) ed anche una *Myiasi delle piaghe* (1) che possono presentare talora interesse dermatologico. Così pure dobbiamo accennare alla **Myiasi intestinale** (3° gruppo), in quanto è possibile che dal tubo digerente la larva migri e raggiunga la pelle, meccanismo patogenetico questo, che da varii A.A. si sostiene in alcune fra le più tipiche M. cutanee propriamente dette, mentre, in altri casi e più facilmente, l'infestione avviene direttamente dall' esterno.

Ma un interesse veramente speciale hanno per noi le **Myiasi cutanee** propriamente dette (1° gruppo). Esse vengono oggi divise in 3 tipi principali dai maggiori AA., a seconda dell' aspetto e del decorso clinico:

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(1) Appare giustificato il distaccare — come fa BRUMPT — nettamente le M. delle piaghe dalle M. cutanee propriamente dette: infatti le M. delle piaghe — che sono anche da considerarsi come una complicanza di altre affezioni e non come una forma a sè, primitiva — interessano quasi sempre altri e più tessuti all' infuori della pelle. Nella tavola riassuntiva annessa non sono quindi ricordate le specie parassite delle M. delle piaghe per le quali rimando il lettore, desideroso di maggiori particolari, al lavoro del PORTA ed alla bibliografia di esso.

1° Tipo. - Caratterizzato da un aspetto lineare dell' affezione, che progredisce via via, con andamento per lo più capriccioso, a zig-zag, accompagnato da prurito. È questa la forma nota sotto varie denominazioni tra cui la più usata è quella di " *Creeping disease* „. Brumpt usa ancora la dizione *Myiase rampante sous-cutanée* che è impropria, perchè la sede dell' affezione non é affatto sempre nel sottocutaneo.

2° Tipo. - *M. sottocutanea a tumori ambulanti*, o a tumori periodici di Hoeg, talora a lungo decorso, con formazione di tumori infiammatorii collegati fra loro da un piccolo cordone d' infiltrato, che segna la via seguita dalla larva, mentre i detti tumori segnano le tappe di arresto.

3° Tipo. - *M. furuncolosa*, nella quale il tumore infiammatorio è la prima ed ultima manifestazione provocata dalla specie parassita. Può quindi esser chiamata anche meglio " M. a tumore infiammatorio unico „.

Abbiamo dunque visto sotto quali aspetti clinici i Ditteri C. S. agiscono da Dermatoendozoi, ci resta da accennare quali siamo fra i numerosissimi Ditteri quelli che presentano tale comportamento. Brevemente dirò che due sott' ordini dei Ditteri C. S. ci interessano:

1° quello dei ***Muscoidi*** Calipteri con due famiglie (fam. **Sarcofagidi** e fam. **Muscidi**)

2° quello degli ***Estroidi*** con la sola famiglia **Estridi** per le due sottofamiglie ***Estrini*** e ***Cuterebrini.***

Assai numerosi sono i generi e le specie qui raccolte, e molte di esse sono citate con varie denominazioni dai vari Autori. Nella tavola riassuntiva qui acclusa, per evitare confusione, questi generi e queste specie sono quasi sempre nominati con le denominazioni consacrate dalla autorità del Brumpt (vedi tavola riassuntiva degli Artropodi Dermatoendozoi).

Ma, fin'ora, nella trattazione degli Insetti Pterigoti ci siamo occupati di uno solo degli ordini in cui essi si dividono, quello dei Ditteri. Ora, altri ve ne sono interessanti dal punto di vista del dermatoendozoarismo.

Sono note infatti nell'ordine ***Afanitteri,*** famiglia **Sarcopsillidi**, le specie: *Sarcopsylla penetrans*, *S. gallinacea*, *S. canis*. Di esse la prima è specialmente nota ai cultori di parassitologia tropicale. (Da notarsi che di queste specie sono parassiti gli adulti e non le larve).

E ancora un terzo ordine, oltre i Ditteri e gli Afanitteri, noi dobbiamo ricordare, quello degli ***Imenotteri,*** per la famiglia **Formicidi** e cioè in seguito alla recentissima descrizione di Klander ed Hollingsworth di un caso di Creeping disease dovuto ad una Formica del genere *Solenopsis*, caso, però, che dobbiamo forse ritenere come del tutto accidentale.

E infine, sulla base dei due casi che esporrò diffusamente, mi è stato possibile dimostrare che vi sono dermatoendozoi anche in un quarto ordine, quello degli Emitteri. Infatti le larve che ho raccolto appartengono ad Insetti dell'ordine ***Emitteri,*** e più precisomente del sott'ordine ***Omotteri,*** famiglia **Cóccidi**, sottofamiglia ***Lecanini.***

Abbiamo dunque potuto vedere nell'insieme tutti quegli Atropodi Tracheati che, allo stato attuale delle conoscenze, si comportano da dermatoendozoi. Ma vi sono altri animali che presentano tale comportamento.

Infatti, lo studio della letteratura su una delle più rare interessanti caratteristiche forme di dermatoendozoarismo, la cosidetta Creeping disease, ci obbliga a riconoscere per dermatoendozoarie anche alcune specie di Elminti.

Gli Elminti dermatoendozoi.

Leggiamo infatti che in Giappone ed in Cina, in forme clinicamente affatto simili alla classica Creeping disease da larve di insetti, sono state trovate forme adulte di Nematelminti del genere *Gnathostoma*, e più precisamente delle specie *G. hispidum* (Morishita, Morishita e Faust) e *G. spinigerum - seu siamense* - (Tamura, Dohi, Sakai). Inoltre Deuntzer e Lewinsen dimostrarono lo stesso *G. spinigerum*, non in una Creeping disease, ma in un isolato tumore infiammatorio del sottocutaneo. E ancora, Ikegami Yutaka, in un caso pure a tipo Creeping disease, trovò una forma diagnosticata per *Echynorhynchus sphaerocephalus*.

E infine, in America, Kirby-Smith, Dove e White, che osservarono un enorme numero di individui affetti da una forma di Creeping eruption con caratteri un po' speciali (facile molteplicità delle lesioni a tragitto, possibilità di fatti generali come dimagramento ed anemia secondarii), 5 volte su 200 biopsie hanno dimostrato un verme parassita per il quale hanno creato il genere provvisorio "*Agamonematodum*". Questo loro *Agamonematodum migrans* fu osservato e descritto in base a reperti microscopici ottenuti con sezioni seriate delle dette biopsie, che furono prelevate agli estremi di tragitti scavati nella pelle.

Le specie di Elminti sopra citate non sono le sole che presentino fatti di dermatoendozoarismo. Altre ve ne sono, e molte, le quali non presentano, è vero, il quadro clinico caratteristico delle precedenti (e cioè assai vicino alla Creeping disease vera, da larve di Insetti), ma esplicano esse pure la loro azione parassitaria per la maggior parte nel tessuto sottocutaneo, producendo talora dei quadri clinici che molto si avvicinano alle altre due manifestazioni che, oltre la Creeping disease, studiammo a proposito delle Myiasi cutanee, voglio dire la forma a tumori ambulanti e la forma a tumore infiammatorio isolato.

Scorrendo il già citato manuale del Brumpt, possiamo vedere numerose specie a noi interessanti per dermatoendozoarismo: ricordiamo tra i **Platelminti**.

— la *Fasciola epatica* che può, talora, embolizzare un vaso periferico e dare, secondariamente, una forma ascessuale del sottocutaneo. Questa specie appartiene ai *Trematodi*, vediamo ora fra i *Cestodi:*

— La larva della Tenia solium, il *Cysticercus cellulosae* che può dare interessanti manifestazioni cutanee (¹),

— L'*Echinococcus granulosus* che può dare non rarissimamente cisti idatidee cutanee.

— Fra i Botriocefalidi, la larva di *Diphyllobothrium Mansoni* fu riscontrata come dermatoendozoaria da Ijima e Murata, Sambon, Daniels (sempre fuori d'Europa), e così pure lo *Sparganum prolifer* che in numerosi esemplari si incista nel sottocutaneo, tanto che Kodama afferma che talora ivi se ne possono contare centinaia per decimetro quadrato. Il caso di Tashiro, tra l'altro, presenta un tipico decorso a tumori ambulanti.

E ancora, fra i **Nematelminti** ricordiamo:

— L'*Enterobius vermicularis* (*Oxyuris verm.*) che « in certi casi troviamo in abbondanza nelle lesioni da lui prodotte sotto l'epidermide del perineo o del solco genito-crurale » (Brumpt),

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(¹) Vedi il recentissimo lavoro di Truffi: " Sulla Cisticercosi dell'ipoderma „ in Arch. It. di Derm. e Ven., 1928, f. III.

— Lo ***Strongyloides intestinalis***, l'***Ancylostoma duodenale***, il ***Necator americanus*** presentano un particolare dermatoendozoarismo — di passaggio, per così dire — in quanto attaccano l'uomo per lo più penetrando attraverso la pelle. Ora si può fare l'obbiezione — che vale anche per molte delle specie prima accennate e che accenneremo in seguito — che il dermatoendozoarismo che osserviamo in loro non è che il particolare di un quadro prima o poi generalizzato, con la partecipazione comunque di altri e più organi. La constatazione non infirma il fatto, per noi interessante, della presenza del parassita dentro la cute o dentro il sottocutaneo, ove può dare manifestazioni morbose tipiche, tanto più che qualche cosa di simile vediamo, ad esempio, nelle Myiasi (vedi forme cutanee contemporanee o susseguenti a M. cavitaria o intestinale).

— Così pure lo studio delle **Filarie** ci offre di interessante le varici cutanee linfatiche zeppe di *Wucheria Bancrofti* i tumori sottocutanei da *Onchocerca volvulus* (« che — anche — può invadere il derma situandosi negli strati superficiali di esso, in pieno connettivo e sempre all'infuori della rete vascolare » Brumpt), e la discussa questione della « scabbia da filarie ». E ancora l'*Onchocerca caecutiens* (Guatemala) che vive in tumori sclerosi sottocutanei e può dare una Onchocercosi cutanea, a forma acuta, erisipeloide, e a forma cronica, indurativa, col colore verdastro caratteristico. Infine la *Loa Loa*, che predilige il cellulare sottocutaneo e vi compie traslazioni rapide e lunghe (producendo edemi fugaci, spesso pruriginosi) durante le quali si possono vedere, ove la pelle è sottile, per trasparenza i contorni del verme:

— Il *Dracunculus medinensis* ben noto per la caratteristica affezione che produce, è pure un endozoo del sottocutaneo.

— E ancora sono da citare il *Gongylonema pulchrum* (¹) trovato agli Stati Uniti (Corvington e Ward) sotto la mucosa del labbro inf. di un ragazzo di 16 anni, il *G. labiale* trovato da Pane in una pustola del labbro superiore di un napoletano, il *G. subtile* descritto da Alessandrini. Viveva nell'epitelio boccale di una persona abitante a Roma (Brumpt).

— E da ultimi ricordiamo ancora i generi Gnathostoma e Agamonematodum, sui quali ci soffermammo già ampiamente.

Riassumendo, numerose sono le specie animali dermotoendozoarie, e sono in parte comprese fra gli Elminti, in parte comprese fra gli Artropodi. A queste ultime noi vogliamo portare il contributo di forme fin'ora non descritte come parassite, e ciò in base ai seguenti

CASI PERSONALI :

CASO I. — Il 25 luglio 1926 si presenta al nostro Ambulatorio tale N. M., contadina, di anni 52, da Garlasco. È una donna abbastanza robusta, in discrete condizioni di sanguificazione e di nutrizione, maritata, con numerosa prole. Lavora sempre in campagna. Nulla presenta d'importante all'infuori della lesione cutanea che l'ha condotta da noi. Ci racconta che da circa due settimane soffre di modico prurito alla fronte ed alla guancia sinistra, talora con lievi sensazioni subbiettive di puntura e di bruciore. Nei primi giorni, quando questi fenomeni erano meno accentuati, essa non riscontrava alterazioni visibili della cute del viso: è da notarsi

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(¹) È interessante il rilevare che queste specie sono vicinissime al *G. neoplasticum*, che Fibiger ha dimostrato essere cancerogeno nello stomaco e nella lingua del *Mus norvegicus*.

tuttavia che essa dice che si osservava con poca attenzione. Dipoi si accorse di una linea rilevata (in corrispondenza della quale avvertiva il prurito) e che andava di giorno in giorno avanzando e dalla tempia destra, donde era partita, contornando la fronte, scendeva verso la guancia sinistra. Infatti, esaminandola, si nota:

L'unione del terzo laterale con il terzo medio del sopracciglio destro segna il punto di partenza di una linea leggermente rilevata che sale sulla fronte, ondulando ora più ora meno, fino quasi alla inserzione dei capelli, poi spostandosi un poco più in basso e lateralmente, contorna quasi tutta la fronte. Dopo averla traversata così quasi compiutamente, scende in basso ed un po' lateralmente arriva quasi all'estremo esterno del sopracciglio sinistro, si perde in mezzo ad esso, riprendendo poi più francamente il suo decorso sulla guancia sinistra, a semicerchio: termina infine all'altezza della linea che congiunge l'ala del naso con il lobo dell'orecchio sinistro (Fig. 1 e 2). Questa traccia lineare, della lunghezza di 20-22 cm. circa, leggermente rilevata, è di colore giallo un po' grigio rispetto al colore della faccia, è accompagnata per la massima parte da un evidente aloncino eritematoso qualche poco desquammante, un po' più evidente al disopra del sopracciglio sinistro francamente accentuato in corrispondenza del semicerchio descritto sulla guancia sinistra.

Alla palpazione si sente un rilievo appena accentuato, indolente più accentuato in corrispondenza della prima e più ancora della seconda sede sopra citate.

L'ammalata ci dice di aver applicato delle pomate (quali non sa precisare) e d'aver fatto un paio di pennellature con tintura d'jodio sulla regione, ma sempre senza alcun risultato.

Davanti all'aspetto caratteristico della lesione, una diagnosi immediata non si potè fare; ci parve doveroso anzitutto di fare esami per allontanare un primo sospetto — che, pur contraddetto da alcuni dati obbiettivi, aveva in favore la figurazione, l'eritema, la lieve desquammazione — che si trattasse cioè di una forma di tricofizia eritemato-squammosa: cominciammo adunque a fare ricerche grattando con un bisturì la linea festonata, raccogliendo il materiale su un vetrino, trattandolo poi con potassa caustica al 40 %, ed esaminandolo microscopicamente.

Dopo una diecina di esami sotto ogni aspetto infruttuosi, in un preparato ottenuto dall'estremo distale (a sinistra) del tragitto, avemmo la sorpresa di trovare un parassita, grande all'incirca come un comune acaro della scabbia, che, fin dai primi esami ci stupì per il suo aspetto che non corrispondeva a nessuna nè delle forme a noi note, nè di quelle riportate nei testi di parassitologia. Insistemmo nei prelevamenti di materiale e senza risultati; infine dovemmo abbandonare la ricerca per intolleranza dell'ammalata. La invitammo a tornare, ma essa non si fece, fino ad oggi, più viva.

Rispetto alla forma clinica ci orientammo presto verso una così detta Creeping disease, con localizzazione probabile del canalicolo sottocornea.

Del parassita per allora non identificato, fu fatto uno schizzo (¹) che riproduciamo tal quale (Fig. 3).

La nostra incertezza sulla classificazione del parassita ed anche sul suo valore come agente causale dell'affezione sarebbe probabilmente rimasta se non ci fosse occorso, un anno dopo, un secondo caso dissimile clinicamente, ma dovuto ad analogo parassita, che, meglio studiato e documentato, ci fece giungere a conclusioni molto importanti.

CASO II. — Il 2 luglio 1927 si presenta al nostro Ambulatorio certo F. N., di anni 25, da Gropello Cairoli, il quale esprime il sospetto di essere colpito da scabbia. Infatti l'esame clinico (come vedremo meglio in seguito), specie per la presenza di cunicoli tutt'affatto tipici per forma e sede, ci fa confermare, ad un primo esame, tale sospetto. Ad ogni modo si preleva con un rasoio una lamella (nostra solita tecnica nella scabbia) di tessuto corneo comprendente un cunicolo, situato nello spazio tra dito indice e medio della mano sinistra. Si monta in potassa caustica 40 % e si osserva

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(¹) Ringrazio vivamente il collega ed amico Prof. Franco Flarer che gentilmente mise a mia disposizione tale disegno, eseguito di sua mano, nonchè le fotografie ed i dati che aveva raccolti su questa interessante ammalata.

al microscopio: non si trova alcun che di parassitario. Dato l'aspetto delle manifestazioni cutanee senz'altro si consiglia l'ammalato ad entrare in Clinica, per un ulteriore accertamento diagnostico e per le cure del caso, ciò che egli promette di fare il domani stesso. Entra invece la mattina del 4 luglio, cioè due giorni dopo: le manifestazioni cutanee sono allora all'incirca quelle osservate al primo esame, forse un poco attenuate.

Trascrivo ad ogni modo la storia clinica compilata il giorno 4 luglio stesso:

" Nulla nel gentilizio; nato di parto fisiologico a termine, ebbe allattamento mercenario, durante il quale, per irrazionale alimentazione, ebbe qualche ritardo — pare – nell'ossificazione. Si rimise poi completamente. Non ricorda d'aver avuto, nel corso del tempo, alcuna malattia degna di importanza, tranne una forma di blenorragia acuta, sofferta lo scorso anno e guarita, dice, in 3 mesi. Non altro. È modico bevitore, fortissimo fumatore. Da notarsi che esercita il piccolo commercio nei cascinali e nei paesi, perciò passa continuamente dall'uno all'altro di essi, camminando assai spesso in mezzo ai campi e sempre calzato di scarpette basse. Inoltre è costretto, dice, a dormire nei luoghi più disparati, non sempre puliti, cambiando di letto quasi ogni giorno.

Fa rimontare l'inizio della forma cutanea attuale a circa 10 giorni prima, quando avvertì la prima senzazione di prurito. Cominciò alle gambe, salì alla cintura, poi via via fino al petto. Contemporaneamente alle gambe, il prurito si fece sentire vivo alle mani (specie negli spazi interdigitali) e subito dopo anche alle braccia. *Non ha fatto alcuna cura fino ad oggi.*

All'**esame clinico** si osserva: Individuo robusto, con muscolatura sviluppata e tonica, pannicolo adiposo scarso, cute abbronzata nelle parti scoperte. Nonostante una certa eleganza degli abiti, è in istato di rilevante sporcizia ed emana pessimo odore. Al capo: nulla all'infuori di una faringite cronica, probabilmente da fumo. Alla stessa causa sono certo da riferire i lievi fatti bronchiali che si riscontrano all'esame del torace. Il cuore nulla presenta clinicamente: ha facile tachicardia emotiva. Nulla all'addome. Sistema ghiandolare linfatico: qualche piccola ghiandola dura, indolente alle regioni laterali del collo ed agli inguini. S. R. W. — — —; Sachs Georgi — — —; Intradermoreazione alla tubercolina $^1/_{1000}$;

leggero +. Urine: limpide, negative le ricerche dell'albumina, zucchero, indacano e urobilina. La numerazione del globuli rossi e bianchi e la formula leucocitaria sono nei limiti normali.

Esame della cute: Nelle sedi indicate dalle figure 7 e 8 si notano evidenti segni di grattamento inframmezzati da rare piccole papule rosee, della grossezza di un grano di miglio, e da alcune piccole vescicoline a contenuto citrino o torbidiccio. Inoltre, esaminando con più attenzione, anche con la lente, si osservano al pube numerosi caratteristici ftirii e lendini sui peli, nonchè numerosi cunicoli, per lo più situati nelle sedi le più caratteristiche per la scabbia volgare, e cioè: la massima parte negli spazi interdigitali della mano destra, alcuni in quelli della mano sinistra; uno sul margine laterale esterno del dorso di questa mano, ed uno anche, evidentissimo, sul dorso della verga, alla metà. Qualcun altro lo si osserva, con caratteri abbastanza tipici, qua e là in mezzo ai segni di grattamento di altre sedi. Questi cunicoli hanno una lunghezza di 2-3-4 millimetri, decorso leggermente arcuato, appaiono di color nerastro e leggermente rilevati sul piano circostante. Due o tre presentano ad un estremo una piccola vescicola a contenuto sieroso e qualcuno pure una lievissima sottile sfumatura eritematosa; palpando con attenzione si avverte distintamente il tenue rilievo filiforme.

Tutti caratteri, questi, che facevano assomigliare in modo veramente speciale questi cunicoli a quelli della scabbia volgare.

Per accertamento diagnostico, col rasoio (nostra consueta tecnica nella scabbia) si preleva, con taglio tangenziale, un cunicolo (il primo, in basso, della Fig. 9), si monta in potassa caustica al 40 %, e si esamina al microscopio.

Con nostra grande sorpresa vi troviamo (¹) otto forme, della grandezza all'incirca di un comune acaro della scabbia, ma con caratteri morfologici tutt'affatto speciali, che non ci fu possibile riportare ad alcuna forma a noi nota. Il reperto curiosissimo ci spinse

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(¹) Tanto in questo caso che nel precedente, nei preparati microscopici si notavano, oltre i parassiti, grande quantità di cellule cornee dissociate ed in lamelle. Si può quindi pensare, — anche in assenza dell'esame istologico (che, come dirò, non fu potuto eseguire) — che la sede dell'affezione fosse sottocornea, come anche facevano già pensare i dati clinici obbiettivi.

ad intensificare le ricerche, nonostante l'intolleranza dell'ammalato, ed ottenemmo buoni risultati.

In tutto furono prelevati diciotto cunicoli, dei quali 7 od 8 da varie sedi (braccia, fianchi, natiche), gli altri dalle mani.

In quattro cunicoli — quelli dimostrati dalle figure 9 e 10 — si ebbe reperto di parassiti, e precisamente:

N. 8 (come dissi) nel cunicolo inferiore dello spazio interdigitale fra medio ed anulare della mano destra;
N. 6 in quello immediatamente sovrastante.
N. 4 nel terzo cunicolo, che sta un po' più in alto, sulla faccia mediale del dito anulare della mano destra;
N. 1 nel cunicolo situato nel margine esterno del dorso della mano sinistra.

Non fu possibile prelevare, per rifiuto dell'ammalato (che era pensionante in proprio), il cunicolo sito sul dorso della verga, nè fare una vera e propria biopsia come era nostra intenzione.

In tutto dunque furono trovati N. 19 esemplari, fra loro del tutto simili, tranne due che — pur presentando tutti gli altri caratteri identici ai precedenti — erano solo un poco più grandi (di un quarto, all'incirca).

Il Prof. Edoardo Zavattari, Direttore dell'Istituto di Anatomia Comparata dell'Ateneo Pavese, nonchè appassionato e profondo cultore di Parassitologia, il quale cortesissimamente si interessò di queste ricerche (¹) e potè di persona assistere e controllare il prelevamento di un cunicolo con reperto positivo di parassiti, diagnosticò trattarsi di larve di Insetti, appartenenti alla famiglia Cóccidi (Cocciniglie) e che non potendo — trattandosi di forme larvali — riferire con sicurezza ad alcuna delle specie

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(¹) Giunga di qui un commosso grazie all'Egregio Prof. E. Zavattari che con sicura dottrina e squisita, affettuosa cortesia, mi ha incoraggiato ed aiutato in questo studio, confortandolo della Sua autorità.

note, abbiamo chiamato provvisoriamente, senza voler attribuire al nome alcun valore sistematico definitivo : " *Dermolecanium migrans* „.

Conseguentemente, la posizione sistematica di questa nuova forma, che presenta notevoli affinità con i ***Lecanini***, viene ad essere stabilita come segue :

INSETTI: ord. **Emitteri**: sottord. ***Omotteri:*** fam. **Cóccidi** : sottofam. ***Lecanini:*** gen. *Dermolecanium*: sp. *migrans*.

Prima di entrare in particolari sulla morfologia dei parassiti, vediamo quale fu il ***decorso clinico*** dell'affezione :

Nel giorno 4-VII-1927 si eseguiscono le ricerche sopra annotate. Nella notte l'a. è preso da febbre alta (40°).

La mattina del 5-VII la febbre è a 39°. Nulla si riscontra all'esame clinico, tranne qualche dolore addominale vago a cui risponde ripienezza di alcune anse intestinali. Risulta dall'interrogatorio che l'a. ha alvo chiuso da più di tre giorni. Si dà purgante salino, lo vomita. Si fa clistere e si ha abbondante emissione di scibale e di liquame fetido. Alla sera la febbre è a 38°.

La mattina del 6-VII l'a. è apirettico. Da rilevare che in questo lasso di tempo, benchè non si sia fatta alcuna cura, la forma è considerevolmente migliorata, sia subbiettivamente che obbiettivamente. Forse anche per questo, tre o quattro nuovi prelevamenti che facemmo furono costantemente negativi. Si depila il pube. Si fa bagno di pulizia, poi, durante i giorni 6-7-8-VII si fanno tre frizioni di pomata antiscabbiosa (Stirace gr. 80, Balsamo del Perù gr. 200, Olio olive gr. 300), poi bagno.

Il giorno 9-VIII una spazzolatura con pomata di Helmerich.

Il giorno 10-VII si fa ancora bagno, si applica pomata emolliente per sedare la lieve reazione prodotta dalle frizioni : il paziente esce di Clinica nella serata e, fino ad oggi, non fu più rivisto.

In possesso di tutti questi dati, volendo trarne una diagnosi, occorreva premettere alcune considerazioni.

Anzitutto un primo sospetto poteva essere avanzato che, (dati anche i caratteri tipici dell'affezione) con la presenza dei Cóccidi potesse coincidere l'esistenza di una scabbia autentica, da sarcoptide comune. Questa possibilità mi pare di poterla escludere:

1° perchè, avendo fatto più di venti prelevamenti di cunicoli, ma ho trovato nè feci, nè uova nè tanto meno acari. Se vi fossero stati non avrebbero dovuto sfuggire ad esami così ripetuti, mentre sappiamo che nei casi di scabbia volgare vergini di cura (come avrebbe potuto essere il nostro) il trovare il parassita o almeno le sue uova o le sue feci è cosa piuttosto facile;

2° perchè nei giorni in cui l'a. per la sopravvenuta febbre stette a letto senza cura (vedi decorso clinico), la forma rapidamente prese ad attenuarsi. Ora, se si fosse trattato di scabbia, il permanere del malato nel caldo del letto avrebbe dovuto (vedi il ben noto prurito notturno degli scabbiosi) portare piuttosto ad un aumento dei fenomeni subbiettivi ed anche del grattamento, e quindi anche dei fenomeni obbiettivi: comunque non mai avremmo avuto un miglioramento;

3° e ancora perchè la larva trovata nel primo caso è — come vedremo meglio in seguito — anch'essa una Coccinìglia. Ora se una larva di Coccinìglia potè da sola provocare una Creeping disease, possiamo ammettere senza fatica che larve di Cóccide abbiano da sole provocata la pseudo scabbia del malato in questione, senza coesistenza nè collaborazione di acari.

Mi pare dunque di poter escludere che vi fossero contemporaneamente Acari in quell'individuo. Ma ancora si potrebbe obbiettare che mentre non è possibile la confusione fra Acari adulti (ottopodi) e Cóccidi (esapodi), potesse trattarsi di larve di acari, la cui prima larva, come è noto, è esapoda. Ora, i caratteri della larva esapoda di acaro (vedi Brumpt, pag. 837 ed in confronto le nostre figure 3-4-5-6) sono tali da rendere impossibile ogni confusione. La presenza nelle larve di Cocciniglia di un rostro e di una segmentazione dell'addome, che mancano nelle larve esapode di acari, basta da sola a farsi che anche alla prima occhiata si debbano distinguere le une dalle altre.

Non è poi da dimenticare che, nel nostro ammalato vi erano numerosi ftirii al pube (le ascelle erano indenni): ora si può affermare con sicurezza che si trattava di pura coincidenza, perchè nessuna parentela vi può essere fra i pediculi ed i parassiti da noi dimostrati. La presenza dei pedicoli può avere interesse per spiegare l'abbondanza di segni di grattamento (vedi Fig. 7 e 8) in alcune sedi (coscie e gambe) in cui vi erano scarsi cunicoli, mentre vi erano alcuni ftirii con numerose lendini attaccate ai peli.

Comunque, a quanto risulta anche dalle sopracitate figure, le localizzazioni cutanee, se non in modo assoluto, erano però ben caratteriristiche (alle mani, per es.) per la diagosi — diciamo così, generica — di « scabbia ». Pur tuttavia preferimmo formulare così la diagnosi: ***pseudo-scabbia da larve di Cocciniglia***: vedemo meglio in seguito il perchè della denominazione pseudo-scabbia.

Non dobbiamo nasconderci tuttavia che vi sono alcune piccole lacune a proposito dello stadio del parassita, dell'attitudine sua, del modo e dei mezzi di attacco rispetto alla pelle. Queste lacune, è bene dirlo fin d'ora, non possono infirmare la realtà incontrovertibile dei reperti ottenuti: di esse poi potremo meglio discutere dopo aver dato, come è necessario, qualche precisione sulle larve trovate e sulla famiglia zoologica cui appartengono. Incominciamo dalla

DESCRIZIONE DELLA LARVA PARASSITA

Dermolecanium migrans Zavattari gen. sp. nn.

Larva. - Ovale allungata, molto simile nella forma generale del corpo alla larva degli altri *Lecanium*. Antenne a superficie scabra di sei articoli, dei quali l'ultimo è ornato di numerosi peli di cui alcuni lunghi quanto la lunghezza complessiva dell'antenna; gli articoli più lunghi sono rispettivamente il sesto e il terzo.

Tre spine stigmatiche brevi, peli lungo i margini del corpo assai lunghi e robusti, ma piuttosto radi. Setole anali molto robuste e molto lunghe così da raggiungere quasi un terzo della lunghezza totale del corpo.

Colore del corpo gialligno-lunghezza totale 400 μ - larghezza massima 215 μ.

Queste larve sono molto simili alle larve di molte specie del genere *Eulecanium* così che la loro distinzione è malagevole e incerta; qui si è ritenuto conveniente dare loro il nome provvisorio di *Dermolecanium migrans*, sia perchè non era possibile una loro sicura attribuzione ad una forma già nota, sia anche per fissare e tenere meglio distinto questo nuovo loro peculiarissimo habitat.

E' tuttavia certo che ricerche ulteriori porteranno con ogni probabilità ad identificare queste larve con larve di specie già note facilitando così lo studio del ciclo biologico di questa interessante Cocciniglia (E. Zavattari).

La famiglia Coccidae si presenta molto interessante dal punto di vista dell' entomologia agraria, infatti i Cóccidi, più conosciuti sotto il nome italiano di Cocciniglie (1), presentano un gran numero di specie, per la massima parte nocive a moltissime piante di grande valore ed utilità. Pur tuttavia fra esse ne troviamo alcune poche da considerare utili, perchè indirettamente ci danno prodotti di pregio. Fra queste ricordo l'*Ericerus pe-la*, dal quale in Cina si ricava cera per candele, il *Kermococcus vermilio* che dà una sostanza colorante rossa, una volta molto usata, la *Tachardia lacca*, che vive in Oriente e produce una lacca nera, d'uso industriale, la *Gascardia madagascariensis* che produce la « lacca del Madagascar » rossa, il *Dactylopius coccus* a cui dobbiamo il carminio che usiamo in istologia, e la *Gossyparia mannifera* (Asia Minore, Egitto) le cui punture attraverso la corteccia della pianta ospite (Tamarix gallica var. mannifera) provocano l'uscita della notissima Manna.

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(1) Vedi la " Monografia delle Cocciniglie italiane „ opera postuma di G. Leonardi, edita — con aggiunta di Appendice — da F. Silvestri, Portici, Stab. Tip. Della Torre, 1922. Vedi anche " Entomologia agraria „ (Manuale sugli insetti nocivi, ecc.) edito dalla R. Sta-

Ma accanto a queste poche specie utili (che vivono fuori d'Europa) ve ne sono tante e tante altre che, in Italia, sono fonte di danni gravissimi per un assai rilevante numero di piante di grande valore. Gli agrumi, molte piante fruttifere dei nostri orti, molti fiori dei nostri giardini, il gelso, l'olivo e moltissime altre piante utili, sono soggette a danni gravissimi, talora irreparabili per l'azione delle Cocciniglie (¹), azione tanto più potente in vista della loro straordinaria fecondità e per il fatto che alcune specie secernono sostanze zuccherine che favoriscono l'impianto di altri pericolosi parassiti, i funghi della « Fumaggine ». Ricordo solo il flagello del gelso, la *Diaspis pentagona*, contro la quale il genio del Berlese, italianissima gloria, scoperse il metodo di lotta della prospaltizzazione.

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zione di Entomologia Agraria di Firenze, sotto la direzione del compianto Prof. Berlese e sotto l'egida del Ministero di Agricoltura, Industria e Commercio, Firenze, Tip. Ricci, 1915.

(¹) Da notarsi che le Cocciniglie non vanno confuse con le *Coccinelle* (dette comunemente da noi *Gallinelle del Signore*, *Bêtes de la Vierge* dai Francesi, *Lady bird* dagli Inglesi) che sono Coleotteri. Le Coccinelle sono nemiche delle Cocciniglie, in quanto ne distruggono gran numero. Così pure i *Cóccidi* (sinonimo di Cocciniglie), non vanno confusi con i Coccídii, che sono Protozoi.

Il ciclo biologico delle CoccinIglie è assai semplice. Di norma le femmine depongono un certo numero di uova sotto il loro corpo o in speciali ovisacchi. Dalle uova nascono le larve che abbandonano la loro primitiva sede e migrano nelle varie parti della pianta ove si fissano per nutrirsi.

Dato questo ciclo e dati i caratteri morfologici della forma, mentre ammettiamo senz' altro che l' infestione venne alla pelle dall' esterno, d' altra parte si presentano numerosi quesiti, e precisamente: « A quale stadio preciso pervennero le forme parassite sull' uomo? In altri termini, sull' uomo depose una femmina le uova oppure giunsero larve già sviluppate? E ancora — ed è questa una questione assai importante — come penetrarono nello spessore della pelle? Vi furono, per così dire, inoculate sotto forma di uova, ad esempio, dalla femmina? (e in questo caso come, con che mezzi la femmina potè compiere questa opera inusitata?).

Oppure furono le larve stesse che — arrivate sulla pelle od ivi sviluppatesi da uova depositate — si sono scavate esse stesse il cammino? Contro anche questa ipotesi sta il fatto che esse non presentano visibilmente organi tali da permettere agevolmente un simile lavoro ».

A queste, e ad altre domande che si potrebbero formulare, non posso io, per ora, dare risposta esauriente; esse però, giova ripeterlo, non possono menomamente infirmare il valore del reperto esatto e controllato dei Cóccidi parassiti dermatoendozoi che hanno provocato nel mio secondo caso la forma pseudo-scabbiosa.

Ora, confrontando i parassiti trovati nel secondo caso con lo schizzo della larva trovata nel Caso I (vedi figure), si può con certezza affermare che anche la Creeping di-

sease che in esso primo caso rilevammo era dovuta ad una larva di Cóccide.

E ancora, l'averne potuto in due casi riscontrare le larve come dermatoendozoarie, permette di includere senza altro le Cocciniglie fra i parassiti dell'uomo, fatto non mai dimostrato, a quel che mi risulta (1).

Ma non solo le Cocciniglie non furon mai dimostrate parassite dell'uomo, ma neppure lo furono mai di altri animali.

Infine, un ultimo rilievo s'impone dal punto di vista della sistematica. Più indietro, nel trattare degli Insetti dermatoendozoi, citammo gli ordini Ditteri, Afanitteri ed Imenotteri, che, fino a ieri, erano i soli tre ordini della Classe Insetti che fossero interessanti dal

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(1) Giova ricordare che è stata avanzata la possibilità di parassitismo sull'uomo per un afide (e gli afidi sono vicinissimi ai Cóccidi) a proposito dell'**Atriplicismo** " malattia cutanea che si riscontra in Cina, caratterizzata da edemi subitanei al volto, alle mani ed alla superficie estensoria dell'antibraccio, con possibili disturbi di movimento e di sensibilità localizzati (formicolio, anestesia) e con circoscritta cianosi cutanea, spesso associati ad estese escare od echimosi delle superfici edematose ". Così testualmente la descrivono NEISSER e JADASSOHN nel loro Manuale e pure vi accenna MONTESANO nel suo recente trattato, e dicono che la malattia è prodotta dall'uso dell'*Atriplex littoralis* (una pianta appartenente alle Chenopodiacee) " o più probabilmente da una varietà di afido (un rincote, od emittero che è sinonimo, come il pidocchio delle rose) che vive su questa pianta, e rispettivamente al veleno di esso ". Tuttavia CASTELLANI, nel suo Manual of Tropical Medicine, ed. II, p. 177, descrivendo ampiamente l'Atriplicismo, che dice legato all'*ingestione* dell'*Atriplex littoralis* e dell'*A. angustissima vel serrata*, accenna anche che da MATIGNON verrebbe messo in rapporto con un parassita che vivrebbe su questa pianta e che si avvicinerebbe — per l'azione patogena — all'*Holothyrus coccinella* Gervais (" a mite ", un acaro dunque) che però vive in altri paesi. CASTELLANI dice qui che l'etiologia dell'Atriplicismo è " estremamente dubbiosa ". Ad ogni modo siamo qui in presenza di nozioni quanto mai incerte, che è doveroso ricordare ma che non possono neppure esser avvicinate a quanto è per noi in questione.

dal nostro punto di vista. Rilevammo allora che i nostri casi personali mettevano in campo un quarto ordine, quello degli Emitteri, che era già noto per contenere specie dermatoepizoarie (genere *Cimex*, *Reduvius*, *Triatoma*, ecc., succiatori di sangue) ma nessuna specie dermatoendozoaria.

Ora, è il primo sott' ordine (o degli Eterotteri) che, negli Emitteri, contiene tutte le specie parassite dell'uomo, il secondo sott' ordine invece, quello degli *Omotteri*, non viene considerato fra quelli interessanti la parassitologia umana. Brumpt infatti si limita a dire in una nota (pagina 933) che De Bergevin avrebbe riconosciuto qualche specie di Omotteri accidentalmente ematofaga (dermatoepizoarismo) e che sarebbero necessarie nuove ricerche di controllo su questi fatti piuttosto insoliti.

Invece le nostre constatazioni ci permettono di affermare che anche il sott' ordine Omotteri (in quanto ad esso appartiene la famiglia Cóccidi) contiene parassiti dell'uomo.

Posso ora, riferendomi anche a quanto ho esposto nella prima parte del presente lavoro, esporre la annunciata ***Tavola riassuntiva degli Artropodi dermatoendozoi*** conosciuti, che valga a dare una occhiata d'assieme sullo stato attuale delle conoscenze in merito ed insieme valga ad inquadrare in esse i nostri nuovi reperti :

		Classe		Sottord.		Sottofam.	
ARTROPODI	*TRACHEATI*	**ARACNIDI**	Ordine **ACARI**		Fam. ***(Demodecidi*** (?))		(*Demodex folliculorum var. hominis* (?))
					Fam. ***Sarcoptidi***		*Sarcoptes scabiei* – ***varietà***
		INSETTI	Ord. **EMITTERI**	OMOTTERI	Fam. ***Còccidi***	**LECANINI**	Casi personali – ***Dermolecanium migrans***
			Ord. **AFANITTERI**		Fam. ***Sarcopsillidi***		*Sarcopsylla penetrans* – *S. gallinacea* ***(Daniels)*** *S. canis* ***(Brumpt)***
			Ord. **DITTERI** **CICLORAFI** **SCHIZOFORI**	MUSCOIDI	Fam. ***Sarcofagidi***		*Sarcophaga lambens* – *S. ruficornis* *S. tuberosa*
					Fam. ***Muscidi*** (non ematofagi)		*Cordylobia anthropophaga* – *C. rodhaini* genere *Calliphora* (*Lucilia caesar*)
				ESTROIDI	Fam. ***Estridi***	**ESTRINI**	genere *Gastrophilus* – gen. *Hypoderma*
						CUTEREBRINI	*Dermatobia cyaniventris*
			Ord. **IMENOTTERI**		Fam. ***Formicidi***		*Solenopsis geminata Fabr. var.* ***Fufa Jerd.*** (caso di Klander e Hollingsworth)

Inquadrati così i nostri reperti nel quadro generale della parassitologia umana, occorre ritornare ai nostri casi clinici per qualche considerazione a loro proposito.

CONSIDERAZIONI SUL CASO I.

A parte il parassita eccezionale, il nostro primo caso, dal punto di vista clinico, deve esser fatto rientrare nel campo della così detta “ *Creeping disease* „ (1). Infatti la forma lineare dell'affezione, accompagnata da scarsi fenomeni reattivi e da prurito, la progressività della forma, l'andamento tutt'affatto caratteristico, ondulante, sono tutti sintomi che quadrano assai bene con la diagnosi di Creeping disease, che riceve poi ulteriore conferma dal reperto in loco del parassita.

Ora, scorrendo la letteratura, è facile rilevare che il quadro della Creeping disease, specie dal punto di vista parassitologico (secondo noi il più importante) non è affatto definito. Infatti, la casistica — svariata e di tutti i paesi — risente spesso, quando viene commentata ed interpretata, dell'impostazione tutt'affatto personale data dall'A. e, d'altra parte, il sopravvenire continuo di dimostrazioni di nuovi parassiti fa sì che i tentativi di fare un ordinamento preciso della questione risultino o troppo particolaristici (2) o troppo vaghi e spesso vengano sorpassati subito che fatti.

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(1) Per maggiori particolari sulla “ Creeping disease „, così come per la completa bibliografia, a scopo di evitare inutili ripetizioni, rimando il lettore ai lavori principali di Rille e Riecke, Lapin, Tamura, Kirby-Smith Dove e White, Cazenave e Photinos.

(2) Traccia di questo si vede anche nella varietà dei nomi che la forma ha via via ricevuti. (Vedi l'elenco che ne fa Photinos). Senza entrare nella discussione che vi è sulle denominazioni, noi ci atteniamo e atterremo al primitivo nome, che è anche il più usato e il più caratteristico, di “ Creeping disease „.

Il più recente ordinamento proposto è quello del **Cazenave**. Dopo aver descritto un caso personale, esamina partitamente la letteratura sull'argomento e giunge alla conclusione che sotto il nome di Creeping disease sono raccolti numerosi fatti clinici che sono abbastanza disparati, e nei quali si possono distinguere tre gruppi:

1° ***Forma europea ed africana***, che corrisponde alla primitiva descrizione di R. Lee: malattia rara, a lesione quasi sempre unica, che « pare causata da una larva di mosca, forse di gastrofilo » (!). Questa è la forma, per così dire, classica, della malattia. I generi *Gastrophilus* ed *Hypoderma* sono in verità gli agenti principali. I casi conosciuti superano oggi di parecchio il centinaio.

2° ***Un tipo osservato al Giappone***, ove le ramificazioni che talora presenta il solco fanno pensare che il parassita ritorni sui suoi passi. (È questa la forma, cui già accennammo da *Gnathostoma* e da *Echinorhynchus)*.

3° ***Il tipo americano, osservato soprattutto nella Florida***, che pare ivi frequentissimo (2500 casi visti da Kirby-Smith, 300 visti da White, 100 da Mc Glasson ecc.) e nel quale le lesioni sono multiple (anche centinaia su uno stesso individuo) e compaiono quasi simultaneamente. (È questa la forma da *Agamonematodum*, che, oltre alla grandiosità del quadro cutaneo, può talora dare — come dicemmo — fatti generali).

Enumerati i tre gruppi il Cazenave conclude che ognuno di essi racchiude una malattia diversa tutt'affatto dal punto di vista della causa e che quindi, se il nome di Creeping disease viene conservato, esso deve toccare, per priorità, al tipo europeo, descritto da R. Lee, e non al tipo 3°, come vorrebbero gli A.A. americani. Ora, non si può negar valore alla classificazione sopra esposta concepita sotto un triplice punto di vista — regionale, clinico, parassitologico — tanto più che essa rispecchia anche le tappe che ha fatto la conoscenza della Creeping disease dalle prime descrizioni di R. Lee, dalla conoscenza delle prime larve di Insetti parassite, via via fino alla grande estensione che ha preso il problema con i lavori degli A.A. giapponesi ed americani sulle forme dovute ad elminti.

Ma, d'altra parte, la conoscenza del reperto di Klander e Hollingsworth di una Creeping disease da formica ed il nostro caso personale, ci obbligherebbero già — non essendo possibile far rientrare questi due casi in alcuno dei tre gruppi precedentemente fissati — a creare due nuovi gruppi, e cioè:

4° ***Una forma osservata in America e dovuta ad una formica.*** Si trattava di un ragazzo di 10 anni, che presentava una linea serpiginosa, caratteristica di « larva migrans », con partenza dalla pianta del piede sinistro, che poi saliva per il lato esterno e giungeva al dorso del piede. Dalla parte terminale fu estratto il parassita, classificato da Dove e Rohner per *Solenopsis geminata Fabr. var. Fufa Jerd.* (1). Si tratta qui di un caso solo ma talmente eccezionale per l'agente parassitario (una formica) che si stacca nettamente da ogni altro e che farebbe forse ritenere trattarsi di caso accidentale.

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(1) Klander e H. "A lesion of Creeping eruption produced by an Ant„ in Arch. of. Derm. and Syph. 1927, vol. 16, n. 1, pag. 78.

5° ***Un tipo osservato in Italia, dovuto a larva di Cocciniglia***, eccezionale dal punto di vista del parassita. Anche qui si tratta di un caso solo, il quale tuttavia riceve una conferma indiretta dalla dimostrazione del nostro caso II, differente clinicamente ma etiologicamente omologo.

Ma giova ora notare che in numerosi casi (20 % secondo Lapin) che in base ai soli dati clinici vengono fatti rientrare — in modo certo e a ragione — nelle Creeping disease classiche, il parassita non fu trovato, ed anche nella forma americana (che ha una casistica imponente) esso fu cercato in 200 biopsie, delle quali 48 sezionate in serie e trovato così solo 5 volte. Ora, la rarità relativa dei casi nei quali vien trovato il parassita, fa sì che il criterio parassitologico, il solo che nel modo più assoluto permetterebbe una classificazione precisa ed indiscutibile, non può assisterci che poco. Per questo, dato anche che il criterio regionale non è nè assoluto nè sufficiente e che i caratteri clinici (l' unicità da una parte e dall' altra la molteplicità delle lesioni non è costante nè senza eccezione) sono similari sotto molti assetti, ci parrebbe più opportuno non insistere troppo su divisioni e suddivisioni, che, specie in un campo tuttora allo studio, possono parere per lo meno premature. Pur costringendoci a riconoscere che non è una entità ben definita, meglio è parlare in generale di « Creeping disease » (è questo un nome che si attaglia bene a tutte le varietà dai primi casi noti dovuti a larve di Estridi fino a quello causato da larve di Cocciniglia), ed attendere dall' accumularsi della casistica e dal progredire delle ricerche, l' inquadramento definitivo di queste curiose forme di dermatoendozoarismo.

CONSIDERAZIONI SUL CASO II.

Spesso vediamo usato il termine « scabbia » per forme che la scabbia vera ricordano più o meno davvicino, talora solo per qualche localizzazione e per lo spiccato sintoma prurito. Così sentiamo parlare, ad esempio, di una « scabbia da cemento » di origine non certo parassitaria, di una « gale des épiciers », probabilmente dovuta ad un dermato epizoo (*Glyciphagus domesticus*), di una « scabbia dei fornai », degli ottonai, di una « scabbia da filarie » e così via. Ora è ben evidente che qui siamo ben lontani da ciò che è la scabbia vera, — quella da *Sarcoptes scabiei var. hominis* — e che quindi la dizione « scabbia » è usurpata. Ora a noi invece parrebbe giusto lasciare tale denominazione riservata solo alla forma vera: le altre forme — e tutte — comprese quelle da sarcoptidi di origine animale (tanto più che vedemmo che sono spessissimo semplici fatti da dermatoepizoarismo) dovrebbero avere il nome di pseudo-scabbie. Perciò per il nostro caso II abbiamo usato la dizione « pseudo scabbia da larve di Cocciniglia » benchè la forma da noi osservata avesse dei caratteri che ricordavano la scabbia vera in un modo assai superiore a qualunque altra forma alla quale venga attibuito il nome di scabbia.

Tuttavia ci pare degna di molto interesse l'acquisizione che può esistere una forma di dermatosi pruriginosa con cunicoli numerosi e caratteristici di scabbia vera, con sedi pure in massima parte tipiche e con assai facile reperto di Cocciniglie parassite.

Interessante è pure il fatto che il medesimo parassita ha potuto dare in due casi due quadri clinici diversi, una Creeping dis. da una parte ed una forma a tipo scabbioso dall'altra. Questo può avere importanza da un punto di vista più generale. Infatti:

Nella tavola riassuntiva annessa, nella quale sono raccolti gli Artropodi dermatoendozoi, noi vediamo che — scartato, come è giusto, il *Demodex folliculorum v. h.* — dal punto di vista clinico abbiamo due tipi di manifestazioni.

1° ***Manifestazioni a tipo scabbioso*** (dovute ai soli Sarcoptidi e *Cóccidi* per il nostro caso II.

2° ***Manifestazioni a tipo,*** *diciamo così, di* ***Myiasi cutanea*** (o *forma di Creeping dis.* o *forme a tumori ambulanti* o *forma a tumore unico infiammatorio*) prodotte da tutte le molte forme elencate, ivi compresi i *Cóccidi* per il nostro caso I.

Ecco dunque che i Cóccidi sono il « trait d'union » fra l'uno e l'altro tipo di manifestazioni, servendo a stringere fra loro di nuova parentela — oltre il comune dermatoendozoarismo — gli Artropodi elencati in detta tavola.

Inversamente, la letteratura sulla scabbia ci presenta un fatto che, sotto un certo aspetto, avvicina la scabbia alla Creeping disease. Infatti Darier riferisce che Dubreuilh ha visto su di un piede un solco di scabbia di 4 centimetri di lunghezza. Questo solco, che si può considerare simile ad una piccola forma di Creeping disease, viene così inversamente a stabilire ancora il contatto che già i Cóccidi avevano creato con il loro doppio comportamento.

Ma si può andare andare anche più oltre, in base alle seguenti considerazioni:

Gli Artropodi dermatoendozoi danno — lo abbiamo visto or ora — due manifestazioni principali: la forma a tipo scabbioso ad una seconda che può assumere dunque un triplice aspetto:

1° ***lineare, progressivo, a tipo di Creeping disease;***
2° ***a tumori ambulanti;***
3° ***a tumore unico infammatorio.***

Orbene, anche i Vermi dermatoendozoi presentano — e lo vedemmo — un comportamente che rientra agevolmente nell'uno o nell'altra di queste tre varietà elencate. Vedi per esempio:

1° ***le Creeping disease da Gnathostoma ed Agamonematodum;***
2° ***i tumori ambulanti da Sparganum prolifer*** (nel caso di Tashiro, ad es.);
3° ***i tumori unici infiammatorii da Fasciola, da Cisticerco, da Onchocerca.***

Quindi si può concludere che tutti i dermatoendozoi, siano essi Artropodi ad Elminti, provocano quattro tipi di manifestazioni cutanee: un tipo scabbioso cioè e gli altri tre ora descritti. Ed esistono poi specie che fanno da « trait d' union » fra l' uno e l' altro tipo di manifestazioni, fra l' una e l' altra grande famiglia di animali, venendo così a consolidare il quadro generale dei dermatoendozoi.

CONCLUSIONI

Da tutto quanto abbiamo potuto esporre, sia in tesi generale, sia in rapporto con i casi dimostrati e studiati si possono trarre alcune conclusioni:

— I Metazoi parassiti della pelle possono utilmente essere divisi in due classi: Dermatoepizoi e Dermatoendozoi.

— Questi secondi comprendono Artropodi ed Elminti e presentano nell'assieme un comportamento clinico che può essere ricondotto a pochi tipi.

— Fra i Dermatoendozoi, in base a due casi da noi dimostrati, devono essere d'ora innanzi compresi i Cóccidi, insetti, questi, fino ad oggi non mai dimostrati parassiti — non solo dell'uomo — ma neppure di altri animali. Inoltre:

— La Creeping disease può essere prodotta da una larva di Cóccide.

— Pure da larve di Cocciniglia può essere causata una dermatosi assai simile alla scabbia volgare.

— Il nuovo agente di queste forme viene qui chiamato con il nome di "*Dermolecanium migrans*", denominazione, lo ripetiamo, puramente provvisoria e che non vuole avere alcun significato sistematico definitivo.

(Consegnato alla Redazione il 27-IV-1928).

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Nota. - Ricordo qui, essendo uscita quando il presente lavoro era già in corso di stampa, l'interessante osservazione di G. M. **Garibaldi** " Il primo caso di Creeping disease osservato in Italia „. - (Arch. It. di Derm. Sifil. e Ven., 1928, vol. 3°, fasc. 4°). Si tratta di un tipico caso osservato su una donna in Roma: non ne fu trovato il parassita.

SPIEGAZIONE DELLE FIGURE

Fig. 1. - Aspetto della lesione nel caso 1° (fotografia).

Fig. 2. - Schema della lesione nel caso 1°.

Fig. 3. - Schizzo della larva trovata nel caso 1°, vista dal lato ventrale.

Fig. 4. - Microfotografia di una delle larve trovate nel caso 2°, vista dal lato dorsale, a fortissimo ingrandimento (grandezza naturale circa 400 μ).

Fig. 5. - Microfotografia di una delle larve trovate nel caso 2°, vista dal lato ventrale, a fortissimo ingrandimento (grandezza naturale circa 400 μ).

Fig. 6. - Immagine semischematica di una larva trovata nel caso 2° e vista dal lato ventrale.

Fig. 7-8 - Schemi delle sedi affette nel caso II·

Fig. 9-10 - Schemi dimostranti la situazione dei quattro cunicoli nei quali furono complessivamente trovate 19 larve nel caso II.

TAV. I.

DOTT. R. CASAZZA - *Sui Dermatoendozoi, a proposito di un nuovo parassita.*
(*Creeping disease e pseudo-scabbia da "Dermolecanium migrans"*).

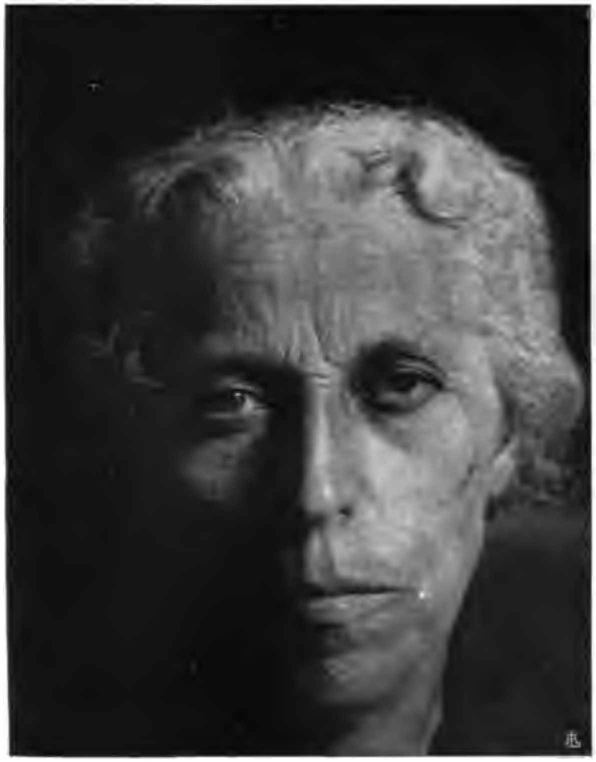

Fig. 1

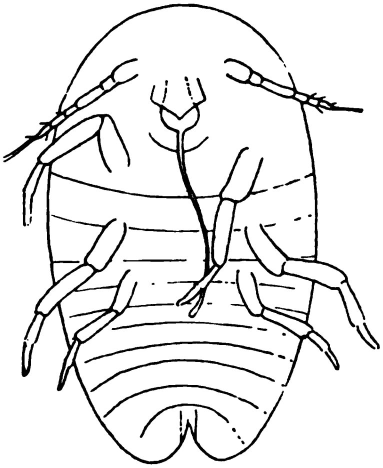

Fig. 3

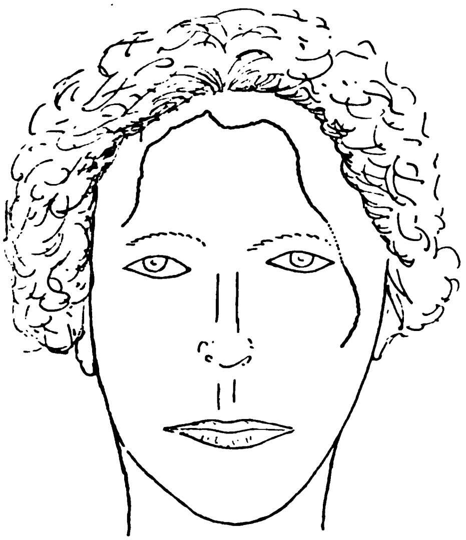

Fig. 2

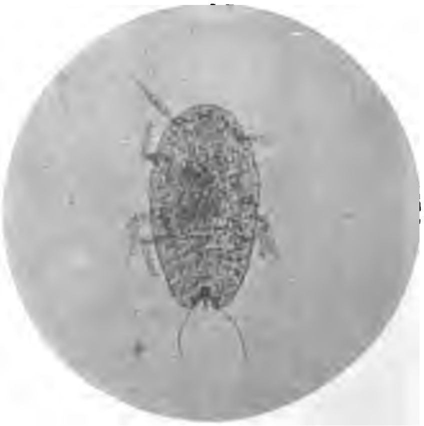

Fig. 4

TAV. II.

DOTT. R. CASAZZA - *Sui Dermatoendozoi, a proposito di un nuovo parassita. (Creeping disease e pseudo-scabbia da "Dermolecanium migrans").*

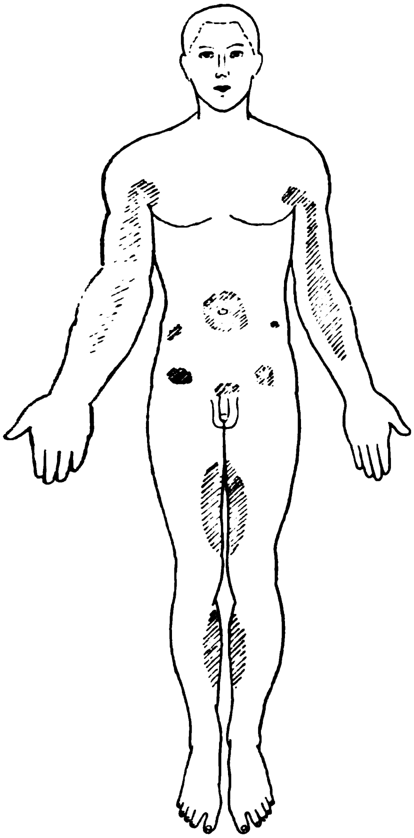

Fig. 7

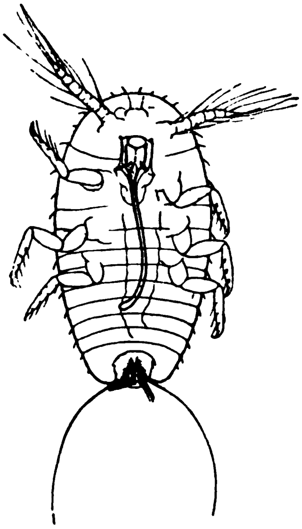

Fig. 6

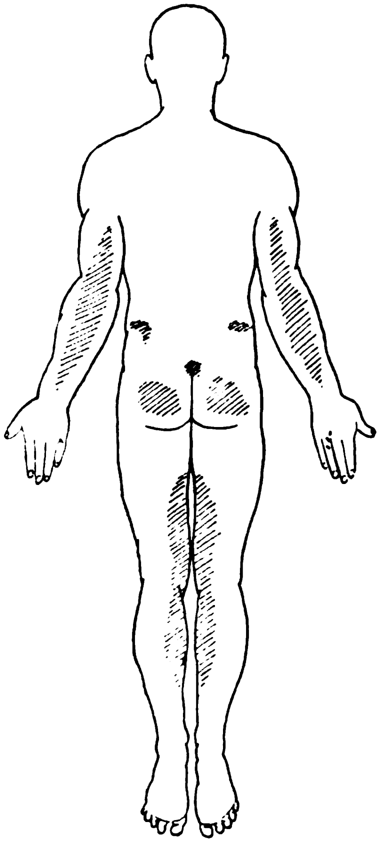

Fig. 8

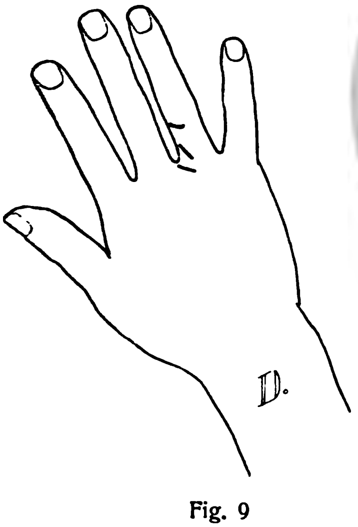

Fig. 9

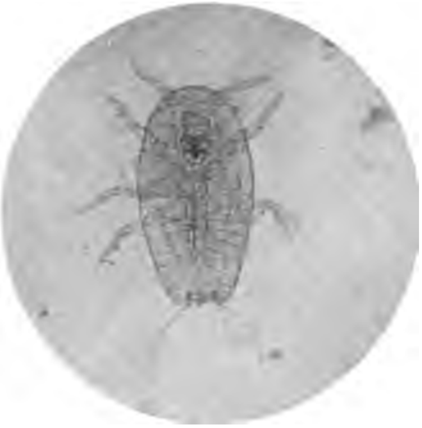

Fig. 5

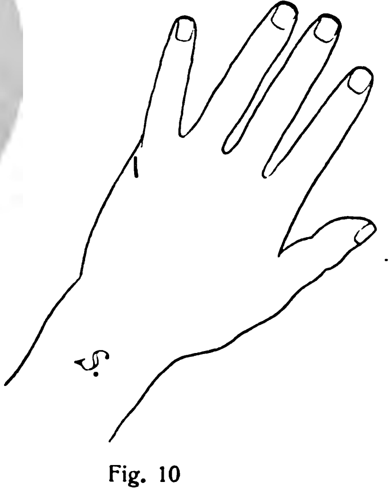

Fig. 10

Intermediate Hosts of Chicken Tapeworms Found in Kansas [1]

A. A. CASE and J. E. ACKERT, Kansas State College, Manhattan, Kan.

The recent findings of Ackert and Case (1938) on effects of tapeworms on chickens have reëmphasized the importance of these parasites. These authors found that cestode infections of even three weeks' duration affected the blood of the chickens and that infections of eight weeks' duration lowered the hemoglobin and sugar content of the blood, and retarded the growth of the chickens. While fowl taeniasis can be checked momentarily by administering taeniacides no effective control of these parasites can be established without breaking the life cycle at some stage. In order to do this knowledge must be gained of the available intermediate hosts. To determine these, life history studies of various species of fowl cestodes have been conducted recently in several laboratories; notably at the United States Zoölogical Division, Washington, at the Veterinary Academy, Hanover, Germany, and at the Kansas State College of Agriculture and Applied Science, Manhattan.

Whereas, a decade ago, garden slugs, earthworms, and flies were thought to be the principal intermediate hosts of chicken tapeworms, the recent findings have shown that ground beetles (Carabidae) are doubtless the principal means of transmitting the more common chicken tapeworms. It was to make known more generally the newer knowledge of intermediate hosts of fowl tapeworms found in Kansas that this study was undertaken.

NORTH AMERICAN CHICKEN TAPEWORMS

Of ten species of tapeworms found in the United States (Cram, 1928), the following six species have been reported from Kansas:

Raillietina cesticillus (Molin, 1858).
Choanotaenia infundibulum (Bloch, 1779).
Raillietina tetragona (Molin, 1858).
Hymenolepis carioca (Magalhaes, 1898).
Raillietina echinobothrida (Megnin, 1880).
Amoebotaenia sphenoides (Railliet, 1892).

The other four species reported from this country are: *Davainea proglottina* (Davaine), *Diorchis americana* Ransom, *Hymenolepis cantaniana* (Pol.), and *Metroliasthes lucida* (Ransom).

KANSAS FOWL CESTODES AND THEIR INTERMEDIATE HOSTS

Raillietina cesticillus

The fowl cestode that probably is of the most common occurrence in Kansas is the rather large species, *Raillietina cesticillus,* whose habitat is the duodenum. Ferry (1934) reported it from sixty-two percent of the chickens he examined. However, Adams and Geiser (1933) found it in only twelve percent of the chickens they examined in Texas. Although houseflies can prob-

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Trans. Kansas Acad. Science, Vol. 42, 1939.

[1] Contribution No. 205 from the Department of Zoölogy, Agricultural Experiment Station, Kansas State College of Agriculture and Applied Science.

ably act as secondary hosts (Ackert, 1918; Reid and Ackert, 1937) the most important intermediate hosts are various species of beetles (Coleoptera), especially those of the family Carabidae (ground beetles). Besides several genera of the families Scarabaeidae, Tenebrionidae, and Ostomidae, there are eight genera and twenty-five species of Carabidae which may serve quite readily as intermediate hosts. The beetles most frequently found at Manhattan (Riley county, Kansas) are of these species. In half an hour in midsummer (July) it has been possible to collect 175 Carabidae on the campus of the Kansas State College. The most numerous of these Carabidae are species of the genera *Cratacanthus* and *Amara*. After a morning rain, beetles of these genera may be seen by the hundreds. Species of *Cratacanthus* and *Amara* have been found to be the best intermediate hosts for the cestode, *R. cesticillus* as determined by experimentation. The ease with which mature cysticercoids can be developed in beetles has led to the use of *R. cesticillus* for experimental studies of Ackert and Case, mentioned above, of Ackert and Reid (1937), who demonstrated an age resistance of older chickens to the growth of these cestodes, and of Harwood and Luttermoser (1938), who found that infections of this cestode retarded the growth of chickens.

Intermediate Hosts of R. cesticillus

Coleoptera: Family Cantharidae: **Podabrus modestus* Say; Family Carabidae: *Amara* sp., *Amara basillaris* (Say), *Amara fallax* Lec., *Amara* (*curtonotus*) *laticollis* Lec., *Amara* (*celia*) *muscula* Say., *Amara* (*Percosia*) *obesa* Say., *Anaferonia* (*Pterostuchus*) sp., *Anaferonia constricta* Say., *Anaferonia* near *substriata* (Lec.), *Anisotarsus* sp., *Anisotarsus agilis* (Dej.), *Anisotarsus subvirens* Csy., *Chalenius tomentosus* Say., *Cratacanthus dubius* Beauv., *Harpalus* sp., *Harpalus faunus* Say., **Harpalus herbivagus* Say., *Harpalus pennsylvanicus* DeGeer., *Pterostichus* near *permundus.*, *Pterostichus* (*Poecilus*) *chalcites* Say., *Pterostichus* near *constricta.*, *Pterostichus* (*Abacidus*) *permundus* Say., *Pterostichus* (*Eumolops*) *torvus* Lec., *Selenophorus* (*pedicularius* DeG.), *Triplectrus rusticus* Say.

Family Ostomidae: **Tenebroides mauritanicus* (L.); Family Scarabaeidae: *Choeridium histeroides* (Web.), *Aphodius granarius* (L.); Family Tenebrionidae: *Tenebrio* sp., *Tribolium confusum* Duval., *Tribolium* sp.

Hymenolepis carioca

Second in the list of tapeworms from Kansas chickens appears to be *Hymenolepis carioca*, a delicate, threadlike cestode which may be present in large numbers (500 or more) in a single bird, according to Ferry (1934). Twiehaus (unpublished) likewise found large infections of this cestode in ailing chickens in Kansas that were sent the Kansas State College Poultry Diseases laboratory in April, 1939. Adams and Geiser found this species to be the most abundant in Texas (37 percent). As to transmission, Cram and Jones (1929) found that various species of Coleoptera may act as intermediate hosts of *H. carioca*. Subsequent studies by Jones indicated that the beetles that could transmit these tapeworms belong to the Scarabaeidae and possibly to the Carabidae.

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* New intermediate hosts of *R. cesticillus* recently announced (abstract) by Case and Ackert (1938) are marked with an asterisk.

INTERMEDIATE HOSTS OF H. CARIOCA

COLEOPTERA: Family Carabidae: *Anisotarsus agilis* (Dej.); Family Scarabaeidae: *Aphodius granarius* (L.), *Choeridium histeroides* (Web.); Family Histeridae: *Carcinops quatuordecimstriata* Steph.

DIPTERA: Family Muscidae: *Stomoxys calcitrans* L.

Raillietina tetragona

The third ranking tapeworm in prevalence in the vicinity of Manhattan appears to be *Raillietina tetragona*. It was third (38 percent) in Ferry's studies in eastern Kansas and second in Texas (Adams and Geiser). This large tapeworm attaches itself to the lower portion of the small intestine and may produce nodules of the intestine which closely resemble lesions of tuberculosis. Horsfall (1938) reported that the common pavement ants *Tetramorium caespitum* (L.) and *Pheidole vinelandica* Forel. could serve as intermediate hosts of *R. tetragona*. Studies upon ants as means of transmitting chicken tapeworms from one host to another are in progress by the writers at the Kansas Experiment Station.

INTERMEDIATE HOSTS OF R. TETRAGONA

HYMENOPTERA: Family Formicidae: *Tetramorium caespitum* (L.), and *Pheidole vinelandica* Forel.

Choanotaenia infundibulum

A fowl cestode that is of very common occurrence in the vicinity of Manhattan, Kan., is *Choanotaenia infundibulum*. Both Ferry (1934) and Adams and Geiser (1933), however, found this species to be the least abundant of the tapeworms they studied. Its habitat is the duodenum.

As to intermediate hosts, Guberlet (1916) found that houseflies (*Musca domestica* L.) are a means of transmitting *Choanotaenia infundibulum* from one chicken to another. Cram (1928), Cram and Jones (1929) and Wetzel (1936) obtained evidence that pointed to beetles as being important intermediate hosts of this tapeworm. Horsfall and Jones (1937) added six species of beetles and two of grasshoppers as new intermediate hosts of *C. infundibulum*.

Further evidence that houseflies may serve as intermediate hosts of *C. infundibulum* was presented by Wetzel (1936), who reported that only twenty percent of the houseflies in his experiments developed cysticercoids and that the number of cysticercoids was small. Reid and Ackert (1937) found a natural infection of ninety-one cysticercoids in a housefly at Manhattan, Kan. Even if only twenty percent of the houseflies developed cysticercoids, the quantities of flies around poultry yards at certain times of the year could easily supply the cysticercoids necessary to produce the heavy tapeworm infections (75 percent) in the naturally infected chickens in an experiment by Reid and Ackert (1937) at Manhattan, Kan.

INTERMEDIATE HOSTS OF C. INFUNDIBULUM

COLEOPTERA: Family Carabidae: **Amara fallax* Lec., **Anaferonia constricta* Say, *Crataeanthus dubius* Beauv., *Stenocellus debilipes* (Say), *Stenolophus conjunctus* (Say); Family Ostomidae: **Tenebroides mauritanicus* (L.); Family

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* New intermediate hosts of *Choanotaenia infundibulum* (Block.) marked with an asterisk. Indebtedness is acknowledged to H. R. Bryson, L. L. Buchanan, and to W. S. Fisher for aid in identifying these beetles.

Scarabaeidae: *Aphodius granarius* (L.), *Aphodius* sp., *Geotrupes sylvaticus* Panz.; Family Staphylinidae: *Apocellus sphaericollis* (Say); Family Tenebrionidae: *Alphitophagus bifasciatus* (Say).

DIPTERA: Family Muscidae: *Musca domestica* (L.).

ORTHOPTERA: Family Locustidae (Acrididae): *Dicromorpha viridis* (Scudder); *Melanoplus femur-rubrum* (DeGeer).

Raillietina echinobothrida

Among the less abundant fowl tapeworms in Kansas is the largest species *Raillietina echinobothrida*. This worm resembles *R. tetragona* closely and is distinguished from it with difficulty. *R. echinobothrida* also attaches itself in the posterior part of the small intestine, and has about the same effects upon the host as *R. tetragona*. Ferry (1934) found this cestode in four percent of the chickens examined in Douglas county, Kansas, and Adams and Geiser found it in twelve percent of the fowls examined in Texas. According to Horsfall (1938), the common pavement ant, *Tetramorium caespitum* may serve as an intermediate host of *R. echinobothrida*. An interesting observation was made by Horsfall (1938), who stated, "The first clue to the intermediate hosts of *R. echinobothrida* and *R. tetragona* was discovered while the writer had under observation in the experimental yard several fecal samples containing *R. echinobothrida* proglottids. An ant carried one of these segments from the feces to an entrance to a nest and disappeared with it. Ants were then examined from this yard and all were found to be negative until August 16, 1935, at which time three *T. caespitum* were dissected and found to contain four cysticercoids the scoleces of which resembled those of *R. echinobothrida*." Larvae of *R. echinobothrida* in the naturally infected ant *Tetramorium semilaeve*, from Marseilles, France, were reported by Joyeux and Baer (1937).

INTERMEDIATE HOSTS OF R. ECHINOBOTHRIDA

HYMENOPTERA: Family Formicidae: *Tetramorium caespitum* (L.), *T. semilaeve* and *Pheidole vinelandica* Forel.

Amoebotaenia sphenoides

A tapeworm present in four percent of the Douglas county, Kansas, chickens examined by Ferry (1934) was the small wedge-shaped *Amoebotaenia sphenoides*. This cestode, which apparently is of rare occurrence in the United States, was reported from eight percent of the chickens examined by Adams and Geiser (1933) in Dallas county, Texas. The intermediate hosts are annelids according to Mönnig (1927) who, in fourteen days, grew the cysticercoids in earthworms [*Ocnerodrilus* (*Ilyogenia*) *africanus*, Beddard]. Four weeks were required for the cysticercoids to develop into adult tapeworms in chickens. Grassi and Rovelli (1889) and Meggit (1916) undoubtedly secured cysticercoids of this tapeworm by feeding the oncospheres to earthworms (*Allolobophora foetida* Eis.).

INTERMEDIATE HOSTS OF A. SPHENOIDES

OLIGOCHAETA: Family Lumbricidae: *Allolobophora foetida* (Eisen), and *Ocnerodrilus* (*Ilyogenia*) *africanus* Beddard.

Davainea proglottina (Not reported from Kansas)

A small tapeworm (four to nine segments) that is found in many countries, but has not been reported from Kansas is *Davainea proglottina*. Because of its minute size it may have been overlooked. Various writers have described it, and Levine (1938) has studied phases of its biology. Wetzel (1936) reported that the most important intermediate host of *D. proglottina* in Germany is the garden slug *Agriolimax agrestis*. Mönnig (1938) lists as intermediate hosts of *D. proglottina* the slugs: *Limax cinerus*, *Arion* sp., *Cepoa* sp., and *Agriolimax* sp., and the snail *Physa heterostropha* (Say). Of these genera, *Agriolomax* and *Physa* are found in Kansas; so that it is possible that *D. proglottina*, for which these gastropods may be intermediate hosts, is also present in Kansas, especially in the eastern and moister part of the state.

INTERMEDIATE HOSTS OF D. PROGLOTTINA

PULMONATA: Family Arionidae: *Arion* sp.; Family Limacidae: *Agriolimax* sp., *Cepoa* sp., *Limax cinerus;* Family Physidae: *Physa heterostropha* (Say).

SUMMARY

1. Of ten species of fowl tapeworms reported from the United States, the following six species have been found in chickens in Kansas: *Raillietina cesticillus* (Molin, 1858), *Choanotaenia infundibulum* (Bloch, 1779), *Raillietina tetragona* (Molin, 1858), *Hymenolepis carioca* (Magalhaes, 1898), *Raillietina echinobothrida* (Megnin, 1880), and *Amoebotaenia sphenoides* (Railliet, 1892).
2. The three following species of beetles are here reported for the first time as intermediate hosts of the chicken cestode *Choanataenia infundibulum: Amara fallax* Lec., *Anaferonia constricta* Say, and *Tenebroides mauritanicus* (L.).
3. The known species of intermediate hosts of the tapeworms found in Kansas chickens consist of beetles, flies, ants, slugs, snails, and earthworms. They are given according to order, family, and genera for the respective tapeworms.
4. Most numerous of these intermediate hosts are ground beetles (Carabidae) of which eight genera and twenty-five species have been identified as intermediate hosts of chicken cestodes.

Altogether nineteen genera and twenty-nine species of beetles may act as intermediate hosts of one or more of the important chicken tapeworms found in Kansas.

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New Intermediate Hosts of Fowl Cestodes[1]

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Recent studies have shown that cestodes have a deleterious effect upon growing chickens (Ackert and Case, 1938; Harwood and Luttermoser, 1938; Levine, 1938). The control of cestode infections requires accurate knowledge of the intermediate hosts of the tapeworms. In a previous paper Case and Ackert (1939) summarized the knowledge of the intermediate hosts of chicken tapeworms found in Kansas. Further work has revealed other intermediate hosts with which this paper deals.

From March, 1936, to March, 1940, over a thousand beetles were studied individually, classified, and their importance as intermediate hosts of chicken tapeworms determined. Most of these beetles were Carabidae (groundbeetles); the remainder were representatives of the families Scarabaeidae (dungbeetles) and Tenebrionidae (flour beetles). The beetles used in the experiments were identified to species by workers in the Bureau of Entomology and Plant Quarantine of the United States Department of Agriculture, Washington, D. C.

In addition to the individual studies, a number of group experiments involving several species of beetles were made. In the group studies, various numbers of beetles of the same genus and species were parasitized experimentally by allowing the insects access to gravid (egg filled) tapeworm segments along with their natural food. Not less than fifteen days were allowed for the eggs to develop into cysticercoids in the beetle; however, many of the beetles carried their infections several weeks. Instead of raising the ground beetles from the egg, vigorous specimens were collected at such distances from any poultry runs that natural infections were not likely. The beetles were kept in moist soil in glass containers as described by Case and Ackert (1939). The cestode species used most were *Raillietina cesticillus* (Molin) and *Choanotaenia infundibulum* (Bloch). Tests involving ants were carried out with gravid segments of *Raillietina tetragona* (Molin).

RESULTS

In the individual experiments, a total of four genera and eight species of beetles not previously reported as intermediate hosts of the chicken tapeworm *Raillietina cesticillus* (Molin) developed cysticercoids to the infective stage. The range of infections was 4 to 129 cysticercoids per beetle with an average of 44. The names of these beetles follow, those marked with an asterisk being additions to the intermediate host list.

Carabidae:
- *Discoderus parallelus* Hald.
- *Pterostichus (Gastrostricta) ventralis* (Say)
- *Agonoderus comma* F.

Trans. Kansas Acad. Science, Vol. 43, 1940.

[1] Contribution No. 215 from the Department of Zoölogy, Agricultural Experiment Station, Kansas State College of Agriculture and Applied Science, Manhattan.

Anisodactylus (*Triplectrus*) *harpaloides* Laf.
Stenolophus conjunctus Say
Scarabaeidae:
Aphodius distinctus (Mull.)
Aphodius cognatus (Lec.)
Tenebrionidae:
Alphitobius diaperinus (Panz.)

Only one beetle not previously reported was found to be an intermediate host for *Choanotaenia infundibulum* (Bloch); it was a groundbeetle, *Lebia grandis* Hentz.

That ground beetles are important intermediate hosts of these tapeworms of chickens is shown by the following observation made upon ten carabids taken from a poultry yard in Reno county, Kansas, by Dr. T. A. Case, who sent them to the writers for examination and classification. Nine of the ten beetles harbored cysticercoids of *Raillietina cesticillus* and four of the nine infected beetles also contained cysticercoids of *Choanotaenia infundibulum*. The mixed infection showed that *R. cesticillus* occurred most frequently by a five to one ratio; the cysticercoids were infective for chickens.

As *Cratacanthus* sp. were plentiful during late spring and early summer, several groups of beetles were placed in fruit jars with moist earth and held at temperatures between 45 and 50 degrees Fahrenheit to determine the possibility of maintaining a supply of beetles during the hot and dry season occurring during late summer and early fall. When needed, the beetles were as lively as when first captured, and lived long enough for the cysticercoids to become infective. No ill results were observed and plenty of beetles were available during a season when a prolonged drought caused the disappearance of nearly all species in the vicinity of Manhattan. About July 1, twenty groundbeetles (*Cratacanthus dubius*) were placed in a refrigerator at 45 to 50 degrees F. and in early November, were removed and parasitized. Seventeen of these beetles lived twenty-one days after eating gravid tapeworm segments and developed cysticercoids which proved infective for chickens.

About eighty percent of a group of cornseed beetles (*Agonoderus comma*) developed cysticercoids after being placed in an environment where abundant small insects were provided as well as gravid tapeworm segments. When individually fed, almost all of these small, flying carabids became infected with cysticercoids of *R. cesticillus*.

Nineteen of twenty confused flour beetles became infected with cysticercoids after being provided with gravid proglottids of *R. cesticillus* in the presence of a mash mixture in which the beetles of this species develop quite readily as pests. The cysticercoids of *R. cesticillus* which develop in the confused flour beetle (*Tribolium confusum* Duval.) are noticeably larger than those that develop in carabids; this may be due to the larger numbers of cysticercoids which develop in carabids. *Choanotaenia infundibulum,* also used this species as a secondary host, development of the infective stage requiring about three weeks.

Six species of ants of common occurrence in this region take proglottids of tapeworms into their nests. Although no cysticercoids have been recovered from the ants, it is quite possible that they are able to act as intermediate hosts in the same manner as those species studied by other workers. Jones and Horsfall (1934) found ants to act as intermediate hosts for *Raillietina tetrogona* and *R. echinobothrida*, two of the largest species of tapeworms found in chickens. Later work by various workers (Joyeux and Baer, 1937; Horsfall, 1938) has established several species of ants as secondary hosts for these two species. Additional research will doubtless reveal a larger number of species as intermediate hosts. Ants[2] known to carry gravid segments of *R. tetragona* into their nests follow:

Formicidae:
  Solenopsis molesta (Say)
  Cremotogaster lineolata var near *cerasi* (Fitch)
  Iridomyrmex pruinosis (Rog) var.
  Pheidole dentata Mayr
  Crematogaster sp.
  Prenolepis (Nylanderia) sp.

SUMMARY

(1) Of over a thousand beetles studied and classified during the last four years, groundbeetles (Carabidae) have predominated as intermediate hosts of two common fowl cestodes, ***Raillietina cesticillus*** (Molin) and *Choanotaenia infundibulum* (Bloch).

(2) Of the 87 species receiving special study during 1939, four genera and eight species proved to be additions to the intermediate host list for *R. cesticillus* and one genus and species was an addition to the secondary host list for *C. infundibulum*.

(3) Nine of ten groundbeetles (Carabidae) collected from a poultry yard at Nickerson, Reno county, Kansas, were infected with cysticercoids of *R. cesticillus;* four had mixed infections with both *R. cesticillus* and *C. infundibulum*. Both species were infective for chickens which likewise developed mixed infections of the tapeworms.

(4) Beetles were successfully kept from one season to another by refrigeration at 45 to 50° F. and were used as intermediate hosts during a season when beetles were scarce in the field.

(5) In several group studies, large percentages of beetles were parasitized with gravid proglottids in the presence of their natural feed.

(6) Six species of ants were observed to carry the ripe segments of *R. tetragona* into their nests. Although no cysticercoids were recovered from these ants, the possibility of their acting as intermediate hosts for this fowl cestode is apparent; various workers have discovered that certain species of ants act as intermediate hosts of both *R. tetragona* and *R. echinobothrida*.

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[2] Determined by M. R. Smith, Bureau of Entomology and Plant Quarantine, Washington, D. C.

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DIRECCIÓN GENERAL DE SANIDAD

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STUDIO DE LAS RATAS Y DE SUS CTOPARÁSITOS EN OCASION EL BROTE EPIDÉMICO DE PESTE EN BARCELONA EN 1931

POR

PABLO CARTAÑA CASTELLA

Y

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MADRID
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1934

COMISION PERMANENTE DE INVESTIGACIONES SANITARIAS
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DIRECCION DE SANIDAD
(PLAZA DE ESPAÑA)
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INTRODUCCION

Iniciada esta encuesta por los doctores Balén y Cartañá, por encargo de la Dirección de la Escuela Nacional de Sanidad, durante el mes de julio de 1931, cuyos resultados han sido publicados recientemente, sobrevino el brote epidémico de peste de agosto a noviembre de 1931. Con este motivo, la Comisión de Investigaciones Sanitarias creyó oportuno su continuación, encargándonos de llevar a cabo un estudio detenido acerca de este tema.

Nuestro trabajo ha sido realizado en la sección de bacteriología y epidemiología del Laboratorio Municipal de Barcelona, a cuyo jefe, Dr. Domingo, así como al director del Centro, Dr. González, nos complacemos en expresar nuestro sincero agradecimiento. Durante su transcurso hemos inaugurado el pabellón de peste y enfermedades exóticas que se ha construído en dicho laboratorio, y que ha contribuído a facilitar grandemente nuestra labor.

Hemos de agradecer también al Dr. Claramunt, director del Instituto Municipal de Higiene, el abundante material que nos ha facilitado, producto de la desratización eficaz que se lleva a efecto bajo su dirección.

Asimismo damos las gracias al Dr. Acosta, director de Sanidad del puerto de Barcelona, cuya colaboración nos ha permitido estudiar los roedores procedentes de la cianhidrización de los buques, y al profesor Dr. Ruiz de Arcaute, cuyas inteligentes enseñanzas nos han sido utilísimas en el curso de este trabajo.

CAPÍTULO I

LOS ROEDORES DOMESTICOS DE BARCELONA

I. Clasificación de los roedores domésticos encontrados.

Las especies de roedores que hemos encontrado durante nuestro estudio en esta capital pertenecen a dos géneros de la familia Múridos, *Epimys* y *Mus,* o sea la rata y el ratón, respectivamente.

Aunque estos dos géneros son fácilmente distinguibles por su talla, pueden diferenciarse más científicamente con arreglo a la siguiente clave adaptada de Cabrera:

1. — Planta del pie posterior con el tubérculo tarsiano interno muy alargado; corona del primer molar poco más larga que la del segundo: EPIMYS.
— Planta del pie posterior con el tubérculo tarsiano interno redondeado; corona del primer molar tan larga, por lo menos, como la del segundo y la del tercero juntas: MUS.

Género EPIMYS TROUESSART.

Sinonimia: *Mus* Linné (part.).
Musculus Rafinesque (part.).
Acanthomys Lesson (part.).
Rattus Fitzinger (no Donovan).

CARACTERES.—La rata se distingue bien de los otros roedores por sus ojos grandes, sus orejas redondeadas, la cola, al menos, casi tan larga como el cuerpo, fina, con apariencia anillada, con pelos muy cortos, poco visibles y escasos. La planta del pie posterior está provista de seis tubérculos, dos anteriores aproximados o reunidos, dos medianos subtriangulares y dos posteriores, de los cuales el externo es redondo y menor que los otros, y el interno es alargado, más del doble de largo que ancho. En el curso de nuestro trabajo hemos podido estudiar ratas pertenecientes a todas las especies y variedades que integran la fauna de nuestro país, y aparte de ellas, alguna que no estaba citada de nuestra fauna, si bien procedía de barcos desratizados en la Sanidad del Puerto. A continuación incluímos una clave que permite diferenciar fácilmente estas especies y variedades.

CLAVE DE ESPECIES Y VARIEDADES DEL GENERO EPIMYS

1. — Cola más larga que la cabeza y el cuerpo juntos, es decir, llevándola hacia adelante por encima del cuerpo del animal, sobrepasa bastante del extremo anterior; las orejas, cuando se las dobla hacia adelante, cubren fácilmente los ojos: E. RATTUS...... 2.
— Cola más fuerte, que, llevada hacia adelante, no alcanza el extremo anterior del hocico; las orejas, más pequeñas, y cuando se las dobla, apenas alcanzan hasta los ojos; hocico más corto y más romo que en la especie precedente: E. NORVEGICUS...... 4.
2. — Pelaje negruzco; el vientre, gris plomizo, con pelos más cortos y adheridos: E. RATTUS RATTUS.
— Pelaje del dorso pardusco o amarillento; vientre blanquecino o gris pálidos... 3.
3. — Cuerpo, por encima, de color pardo grisáceo, que cambia gradualmente a gris más pálido en el vientre; pelaje del dorso más bien áspero, y el del vientre, más largo que en la forma típica: E. RATTUS ALEXANDRINUS.
— Dorso amarillento o pardo leonado; cambia bruscamente en el vientre, cuyo pelaje es completamente blanco o muy levemente amarillento; todo él muy suave, largo y denso: E. RATTUS FRUGIVORUS.
4. — Pelaje del dorso leonado y pardusco mezclados; el vientre, de color blanco sucio: E. NORVEGICUS NORUEGICUS.
— Pelaje negruzco en el dorso y gris obscuro en el vientre, muy parecido al de la rata negra: E. NORVEGICUS HIBERNICUS.

EPIMYS RATTUS (LINNEO): RATA NEGRA.

Sinonimia: *Mus rattus* Linné.
Mus latipes Bennet.
Mus tectorum var fuliginosus Bonaparte.
Mus insularis Waterhouse.
Mus subcoeruleus Lesson.
Rattus domesticus Fitzinger.
Mus alexandrino rattus Fatis.
Mus rattus ater Mallais.

Esta rata, que es muy abundante en los barcos, está, en cambio, muy reducida en la ciudad por la competencia que le hace la rata gris.

CARACTERES.—Su cabeza estrecha y larga, con el hocico puntiagudo, y sus orejas grandes, le dan una fisonomía muy distinta de la rata gris. Su cola es fina y larga, con los anillos muy numerosos, casi desnudos. El pelo del dorso del cuerpo es largo y áspero, mientras toda la región ventral es de color gris de plomo más o menos pálido y con pelos cortos.

Probablemente la presencia de esta rata en Barcelona en una sola casa, que comunicaba con el puerto, es debida a la inmigración de ratas procedentes de barcos, que no han logrado aclimatarse más que en ese punto en el que no hemos encontrado la rata gris.

Por estas razones, creemos que su importancia en relación con la epidemiología de la peste es muy escasa en Barcelona.

Sus dimensiones son siempre menores que las de la rata gris, y el macho adulto tiene unos 185 mm. de largo, mientras que su cola llega a los 200.

E. RATTUS FRUGIVORUS RAFINESQUE.

Sinonimia: *Musculus frugivorus* Rafinesque.
Mus tectorum Savi (part.).
Myoxus siculoe Lesson.
Mus silvestris Pistet.
Mus leucogaster Pictet.
Mus nemoralis Sélys.
Mus picteti Schinz.
Mus alexandrinus Blasius (no Geoffroy).
Mus (Epimys) rattus alexandrinus Trouessart (no Geoffroy).
Mus rattus tectorum Bonhote.

Esta rata parece ser bastante salvaje en tierra, en donde se encuentra sólo en los campos. En cambio, la hemos encontrado con bastante frecuencia en los ejemplares procedentes de la desratización de barcos.

Aunque Cabrera indica que esta variedad ha sido indudablemente transportada por los barcos a algunas regiones del norte de Africa y América, los diferentes autores que hemos consultado no la citan nunca como rata de barco, probablemente por confundirla con el *alexandrinus*.

CARACTERES.—Forma de la cabeza y de la cola como en la especie típica, pelaje del dorso pardusco leonado, más o menos claro y frecuentemente con pelos más claros destacándose sobre los restantes. La parte posterior hacia la cola suele ser más obscura; todos los pelos son más finos que en *rattus* típico. Vientre cubierto de pelos blancos o ligeramente amarillentos, con la base del mismo color. Los flancos están provistos de pelos de esta tonalidad, que contrastan con los del dorso, pero a veces son de un gris pizarra en una faja interna mal definida entre ambas regiones. Sus dimensiones son próximamente las de la forma típica o algo más pequeñas.

En relación con la peste, esta rata no debe jugar ningún papel, al menos en el casco de la ciudad, supuesto que entre los ejemplares de ratas estudiados no hemos podido hallarla. Ahora bien, tratándose de especie campestre, pudiera en algunos casos propagar esta enfermedad en el medio rural de Cataluña, en el cual ha sido señalada su presencia.

E. RATTUS ALEXANDRINUS GEOFFROY.

Sinonimia: *Mus asiaticus* Gray.
Mus arboreus Horfs.
Mus crassipes Blyth.
Mus rattus foss Cornalia.
Mus tectorum Savy (part.).

Esta variedad se encuentra muy frecuentemente sobre los barcos, acompañando a la variedad anterior y a *E. rattus rattus.*

Probablemente se trata de una forma menos fuerte que la *norvegicus,* al igual que la forma típica, por lo cual en la lucha por la existencia ha sido desplazada de la tierra firme. Su abundancia en los barcos en relación con las otras formas varía bastante.

Esta variedad no ha sido encontrada todavía en nuestro país. Sin embargo, nos inclinamos a creer, en contra de la opinión de Cabrera, que debe formar parte de nuestra fauna de roedores, apoyándonos en el hecho de que abundaba en barcos desratizados que hacían simplemente viajes de cabotaje entre diversas regiones de España.

Caracteres.—Forma del cuerpo enteramente igual a la de *rattus rattus;* pelaje del dorso pardo grisáceo y áspero con algunos pelos leonados; este color cambia gradualmente hacia la región ventral, en la cual el pelo es gris pálido y largo. Este tono gris le diferencia de la forma *frugivorus,* con la cual se confundía hasta que el zoólogo inglés Lewis destacó las diferencias constantes entre ambas variedades, que eran sobre todo marcadas en el caso de los pelos del vientre, de un blanco puro en *frugivorus,* y al menos con la base gris en *alexandrinus.*

En virtud de no existir en nuestro país, o al menos de ser rara en él, no ha de desempeñar papel alguno en la epidemiología de la peste.

Sus dimensiones son aproximadas a la de *rattus rattus.*

EPIMYS NORVEGICUS NORVEGICUS ERXLEBEN.

Sinonimia: *Mus norvegicus* Erxleben.
Mus decumanus Pallas.
Mus surmulottus Severinus.
Mus decumanus hybridus Bechstein.
Mus caspius Oken.
Epimys norvegicus Satunin.
Mus (Epimys) norvegicus Trouessart.

Es la especie predominante en Barcelona, de tal modo, que sólo un escaso porcentaje de las ratas examinadas pertenecían a otras formas.

Caracteres.—Cabeza gruesa, con hocico romo, las orejas cortas, que no llegan a tapar el ojo cuando se las fuerza hacia adelante. La cola gruesa y más corta que la cabeza y el cuerpo reunidos, con numerosos anillos y casi lampiña. Pelaje de la parte dorsal de color leonado, con pelos de color canela, cuyo extremo es más obscuro, casi negro, por lo cual la tonalidad en general es más obscura; vientre blanco amarillento grisáceo.

Su tamaño es marcadamente mayor que el de la especie *rattus*, llegando el macho a unos 215 mm. y algo más la hembra adulta, que llega a veces a los 200 mm.

Esta rata goza de un poder de adaptación muy grande, y se ha acomodado a las condiciones más diversas; por ser más fuerte que la rata negra, la ha expulsado de sus antiguas posesiones, reduciéndola sobre todo a los puertos y a los pisos altos de las casas, ya que la rata gris es más amiga de vivir en los sitios a ras del suelo, como almacenes, o por debajo de su nivel, como en sótanos, alcantarillas, etc., siendo considerada en general como buena nadadora. Por el contrario, es menos ágil que la negra, y sus saltos no llegan nunca a ser tan potentes como en ésta.

Aprovechando el canal de aislamiento del pabellón de peste construído en el Laboratorio Municipal, para impedir el escape de los roedores en experimentación, hemos realizado observaciones acerca del poder natatorio de esta especie.

En la mayoría de los casos, las ratas sólo han podido atravesar a nado una distancia de 30 metros aproximadamente, habiéndola cubierto en dos o en tres minutos. Al cabo de ellos ya no avanzan, y sólo hacen esfuerzos desesperados para mantenerse a flote, y son definitivamente vencidas, muriendo ahogadas al cabo de otros dos minutos, o sea que en total no han resistido más de cinco minutos nadando.

Podría oponérsenos que las ratas cogidas en rateras, y que han permanecido en ellas durante, al menos, doce horas, habrán desgastado las suficientes energías para disminuir sensiblemente su potencia de natación; pero, a pesar de ello, creemos demasiado exagerados todos los datos anotados por los distintos autores sobre su vigor. Sobre todo, pensar, como Pallas, que en el año 1727 pasaran legiones considerables de esta especie de ratas hasta las casas de Astrakán atravesando el río Volga, es poco probable, por no decir prácticamente imposible, a no haber utilizado objetos flotantes para reposar.

Esto nos induce a pensar que es más verosímil que hayan sido los barcos provenientes de Rusia los que hayan traído esa plaga a las demás regiones de Europa.

Lo mismo se infiere del estudio de la membrana interdigital, que no es mucho mayor que la de la otra especie.

En relación con la peste, puede decirse que en Barcelona es actualmente la sola especie causante de los brotes epidémicos que se han registrado en la ciudad.

E. NORVEGICUS HIBERNICUS THOMPSON.

Sinonimia: ***Mus decumanoides*** Hodgs.
Mus brunneus Hodgs.
Mus maurus Waterhouse.

Esta variedad tiene una distribución geográfica muy extensa y se encuentra en una serie de países exóticos, habiéndose citado como forma *maurus* en Francia. Trouessart cree que esta forma es un sinónimo de la variedad *hibernicus,* que considera distinta de la especie típica. En un caso u otro, su coloración es bastante, a nuestro juicio, para poder fundar una variedad, con tanto motivo como se ha fundado la *alexandrinus* o la *frugivorus* de la especie *rattus.*

Caracteres.—Muy parecida a la rata negra, por el color de su pelaje, se la reconoce enseguida por la forma del cuerpo y por la longitud de la cola, enteramente igual a la de la forma típica gris.

Pelaje de color obscuro casi negro en su dorso, en el que a veces hay algunos pelos más claros, que no varían el tono fundamental, y de color gris obscuro en su vientre.

Nosotros no hemos podido capturar más que dos ejemplares provenientes de una casa de la ciudad. Estaba citada por Aguilar del Parque de Barcelona, pero parece ser muy rara. Indudablemente no tiene importancia como vector de peste.

Género MUS LINNEO.

Muy parecido a *Epimys,* que algunos autores antiguos incluían dentro de este género. Su tamaño más pequeño y la forma del tubérculo interno tarsiano posterior le distinguen fácilmente en los casos en que, por tratarse de ejemplares jóvenes, pudieran ser confundidos. También ayuda a ello la distinta dimensión de los molares, que en *Mus* son muy distintos y el primero es casi doble que los dos siguientes.

Sólo dos especies en la región catalana, representadas por variedades, que pueden distinguirse con arreglo a la siguiente clave:

1. — Cola casi de igual longitud que la cabeza o algo más corta; pie posterior de más de 17 mm.: MUS MUSCULUS BREVIROSTRIS Waterh.
— Cola visiblemente más corta que el cuerpo; pie posterior de menos de 17 milímetros: MUS SPICILEGUS HISPANICUS Mill.

La única especie capturada por nosotros en las rateras ha sido la primera, pero también está citada de la región catalana la segunda, aunque sus costumbres campestres hacen que se la encuentre rara vez en las casas y almacenes.

Como es sabido, el ratón interviene muy poco en la epidemiología de la peste, aunque es receptivo para ella, probablemente debido a la escasez de sus parásitos. De un modo o de otro, conviene hacer resaltar su nula importancia en la propagación de la enfermedad, a pesar del íntimo contacto en que vive con relación al hombre.

Por esta razón nos limitamos a proporcionar esta clave de especies, con la que puede llegarse a determinar bien de qué ratón se trata, sin describir detalladamente cada una de ellas.

II. Las ratas y sus enfermedades en relación con el hombre y los animales domésticos.

Las siguientes enfermedades pueden ser transmitidas por la rata al hombre o a los animales domésticos que tienen mayor o menor contacto con ella:

PESTE
PSEUDOTUBERCULOSIS
TULAREMIA
SODOKU
TRIQUINOSIS
TIFUS EXANTEMATICO
ESPIROQUETOSIS ICTEROHEMORRAGICA
RABIA
INFLUENZA DE LOS CABALLOS
TIÑA
FIEBRE AFTOSA

Otras enfermedades pueden ser sufridas por las ratas, si bien no son transmisibles al hombre; tales, por ejemplo, una tripanosomiasis producida por el *Tr. lewisi,* y transmitida de rata a rata por la picadura de la *X. cheopis.* La lepra de las ratas, originada por un bacilo idéntico al de la lepra humana, el *B. stefanski,* es padecida también por estos roedores.

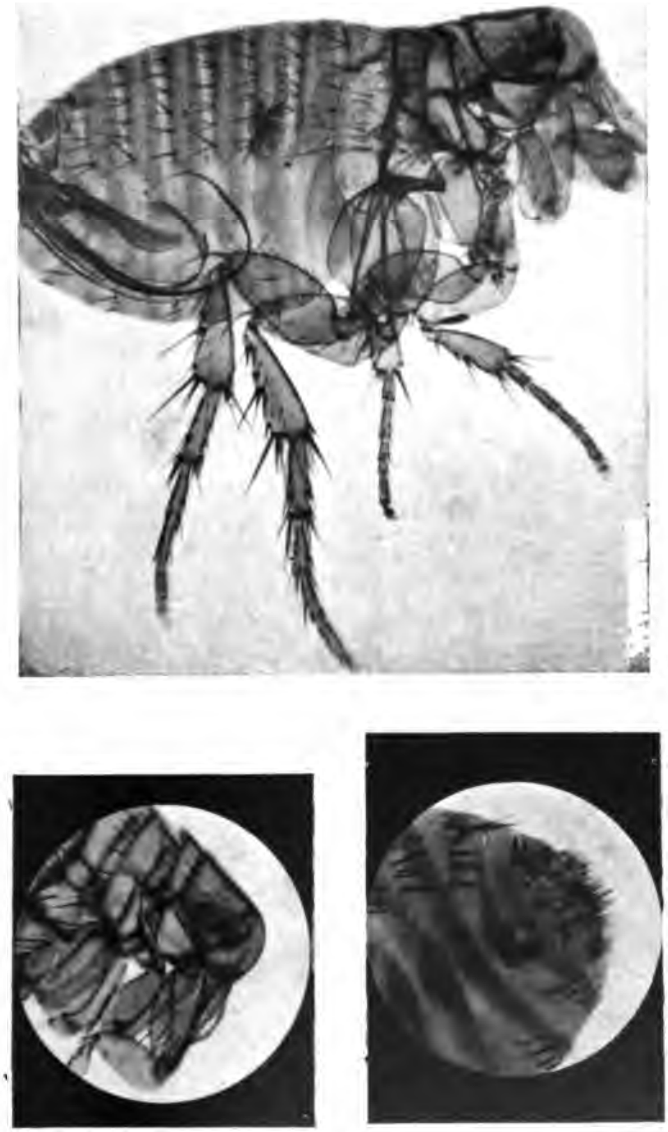
Asimismo sufren una infección debida a la *Bartonella muris,* transmitida por el *Poliplax spinulosus.*

A menudo sufren cáncer de hígado, que se desarrolla alrededor de los quistes de *Taenia taeniformis,* que luego describiremos, así como cáncer de estómago, producido por un pequeño gusano, el *Gongylonema neoplasticum,* que en el estado larvario vive en los músculos de la cucaracha, y que seguramente es adquirido al alimentarse de ellas.

En algunas regiones, entre ellas Barcelona, son parasitadas también las ratas por un Nematodo de la familia de los Tricúridos, el *Hepaticola hepatica,* que se localiza en el hígado, produciendo lesiones que serán descritas más tarde.

El parasitismo intestinal por tenias es frecuentemente muy intenso, lo cual es fácilmente explicable por la alimentación de estas animales, compuesta, como ya indicamos, de detritus, desperdicios, etc. La especie más frecuentemente encontrada es la *Hymenolepis fraterna.* A veces hemos encontrado al realizar la autopsia verdaderos tapones de estas tenias en el intestino de las ratas.

LAMINA 1



Xenopsylla cheopis.—1, macho; 2 y 3, extremo anterior y posterior, respectivamente, de la hembra. La sueropleura está provista del engrosamiento vertical característico de la subfamilia.

(Microfotografías *Vidal-Cartañá.)*

CAPÍTULO II

LOS ECTOPARASITOS DE LAS RATAS DE BARCELONA

I. Afanípteros.

Durante el curso de nuestro trabajo se han observado siete especies de pulgas parasitizando a las ratas de la ciudad, entre las que se encuentran las especies más importantes en la propagación de la peste. La siguiente clave puede servir para distinguirlas:

Clave de especies de pulgas

1.—Cabeza con una muesca profunda en su borde superior, que divide la frente del occipucio, y que continúa el surco antenal; ojos atrofiados: FRACTICIPITOS...... 2.

—La cabeza no está dividida en dos por un surco, siendo su perfil entero: INTEGRICIPITOS...... 3.

2.—Primer segmento abdominal provisto de un peine bien marcado; los restantes segmentos llevan pequeñas espinas; peine genal de 10 a 12 dientes: STENOPONIA TRIPECTINATA.

—Primer segmento abdominal, sin peine; los restantes, sin espinas; peine genal de cuatro púas: CTENOPSYLLA SEGNIS.

3.—Cabeza y tórax, sin peine de espinas...... 4.

—Al menos el tórax, con un peine...... 5.

4.—Cabeza con una serie de cerdas en el borde posterior del occipucio, cuya inserción forma con otras dos cerdas supraantenales una V bien marcada; mesopleura ancha, con un engrosamiento quitinoso central desde la coxa hasta el ángulo superior, y otro desde la coxa al borde anterior: XENOPSYLLA CHEOPIS.

—Occipucio sin cerdas apicales; sobre la antena sólo hay una cerda; mesopleura con un engrosamiento en su borde anterior, que desde él se dirige hasta la inserción de la coxa, sin engrosamiento mediano: PULEX IRRITANS.

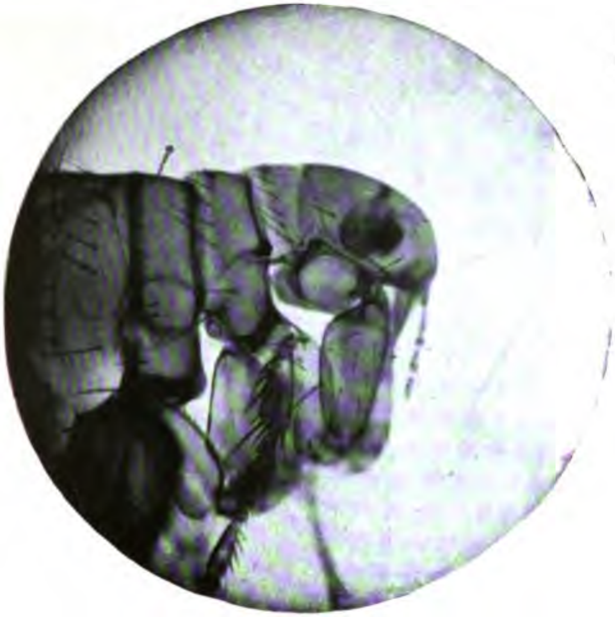
5.—Sólo con un peine protorácico; segmentos abdominales con dos series de espinas, una en el borde posterior, y otra en la parte mediana: CERATOPHYLLUS FASCIATUS.

—Al menos con dos peines, uno en la mejilla y otro protorácico...... 6.

6.—Cabeza corta y con su borde superior redondeado; la parte occipital forma con

LÁMINA 3

1



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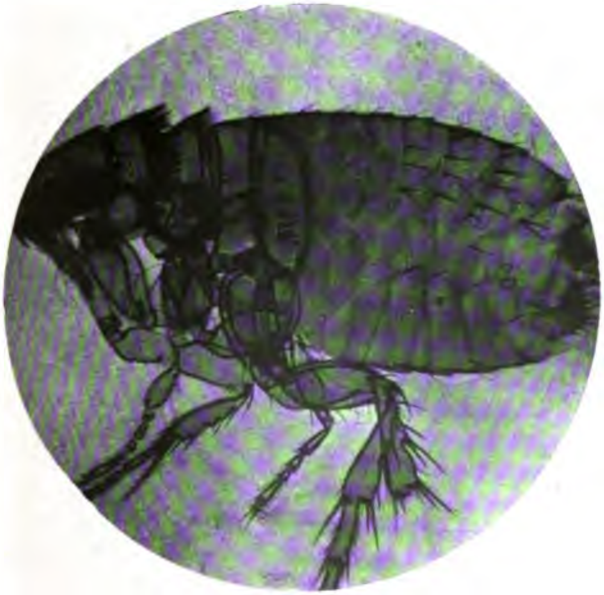


1. *Pulex irritans,* extremo anterior. Nótese la falta de engrosamiento vertical en la sueropleura. 2. *Ctenocephalides felis;* hembra.

(Microfotografías *Cartañá.*)

Género PULEX LINNÉ.

PULEX IRRITANS LINNÉ.

Sinonimia: *¿bovis* Leach?

Es la pulga del hombre y la más rara de las seis especies encontradas más a menudo en las ratas.

Caracteres.—Pertenece también al grupo de pulgas sin peines y con ojos bien desarrollados, como *Xenopsylla,* a la cual se parece mucho. Puede distinguirse de ella fácilmente por tener sólo una cerda supraantenal, por el engrosamiento de la mesopleura, que va desde la inserción de la coxa hasta el borde anterior, sin engrosamiento mediano. Los segmentos abdominales llevan sólo una serie de cerdas medianas, y la espermoteca tiene una cabeza gruesa y una cola bastante más fina que ella, siendo también por eso diferenciable de la *X. cheopis.*

En relación con la peste, se la considera como un vector importante en caso de epidemia, para la transmisión de hombre a hombre.

Género CTENOCEPHALIDES STILES Y COLLINS.

Sinonimia: *Ctenocephalus* Kol.

CTENOCEPHALIDES FELIS BOUCHÉ.

Sinonimia: *Pulex felis* Bouché.
Pulex serraticeps Garv. (part.).
Ctenocephalus ennesdus Kol.
Pulex cati Megnin.
Ctenocephalus serraticeps murina Tir.

Es la pulga del gato, que también puede parasitar frecuentemente a la rata en nuestros climas.

Caracteres.—Cabeza provista de peine en las mejillas, con la primera espina más larga que la siguiente; ojos bien desarrollados; borde superior de la cabeza ligeramente arqueado y formando un ángulo con las mejillas bastante agudo. Antena sólo anillada en su parte superior. Protórax provisto con un peine, de unas ocho espinas grandes a cada lado. Mesopleura con un engrosamiento vertical y otro anterior en la posición del de *X. cheopis.* El macho se distingue de la hembra en que su cabeza es más redondeada y el ángulo que forma en su parte anterior el frontoclipeo con las mejillas es más abierto.

Puede desempeñar el papel de transmisor de la peste únicamente en casos de epidemia.

CTENOCEPHALIDES CANIS CURT.

Sinonimia: *Pulex canis* Curt.
¿Pulex martis? Bouché.
Ctenocephalus novemdentatus Kol.
Pulex serraticeps Gerv. (part.).

No es pulga frecuente en la rata, y se presenta de modo ocasional, quizá debido al contacto de aquéllas con los sitios en que existen los perros.

CARACTERES.—Parecida al *Ct. felis,* se diferencia de él por la cabeza visiblemente menos alargada, por el peine de las mejillas, de espinas más cortas, siendo la primera más pequeña que las siguientes; el perfil de la cabeza es más redondeado y su parte anterior forma un ángulo casi recto con las mejillas. El macho posee los broches más gruesos y mazudos.

Esta pulga, como el *Ct. felis,* puede jugar papel en la transmisión de la peste tan sólo cuando existe una epidemia de esta enfermedad, aunque experimentalmente puede actuar como transmisor.

Género CERATOPHYLLUS CURT.

CERATOPHYLLUS FASCIATUS BOSC.

Sinonimia: *Pulex fasciatus* Bosc (part.).
Pulex furens Dale.
¿Ceratophyllus californicus? Baker.
Ceratophyllus canadensis Baker.
Ceratophyllus oculatus Baker.

Si los caracteres del género *Ceratophyllus* son bastante fáciles de distinguir, no así dentro de este género las diversas especies, que son extremadamente parecidas. *C. fasciatus* es la pulga de la rata de los países templados, y se encuentra extendida por toda Europa. En nuestro país se encuentra no sólo en el litoral mediterráneo, sino también en el interior.

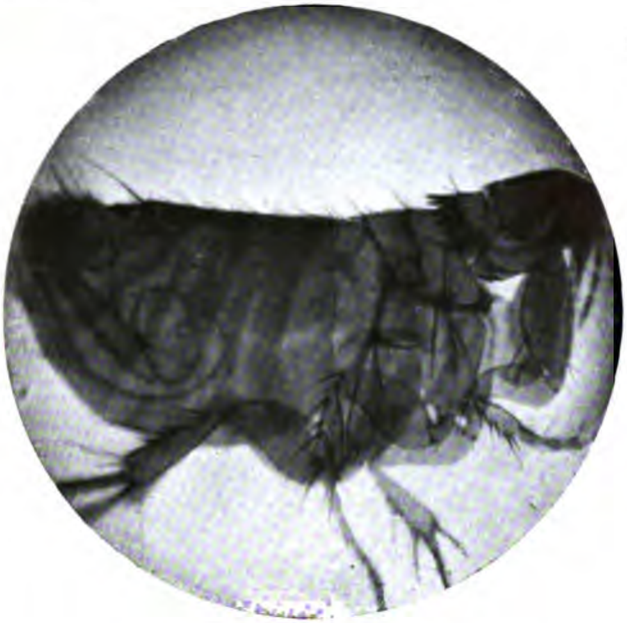
CARACTERES.—Cabeza desprovista de peines, redondeada en su borde superior, con ojos bien desarrollados; antenas con la maza anillada en todo su contorno. Con una sola cerda orbital. Tórax con los escleritos largos y estrechos, el mesoternito con un engrosamiento vertical mediano y su borde inferior también engrosado. Prototórax con un peine compuesto de unas 10 a 11 espinas a cada lado. Espermoteca de la hembra con la cabeza no muy gruesa.

Esta especie se encuentra en considerable número entre las ratas estudiadas, y en los meses fríos sobrepasa con mucho su cifra a la de *X. Cheopis.*

Experimentalmente se ha comprobado que puede transmitir la peste, y probablemente desempeña un papel importante en la propagación de la epidemia en los lugares de clima templado, en los que *X. cheopis* no es muy abundante.

LAMINA 4

1



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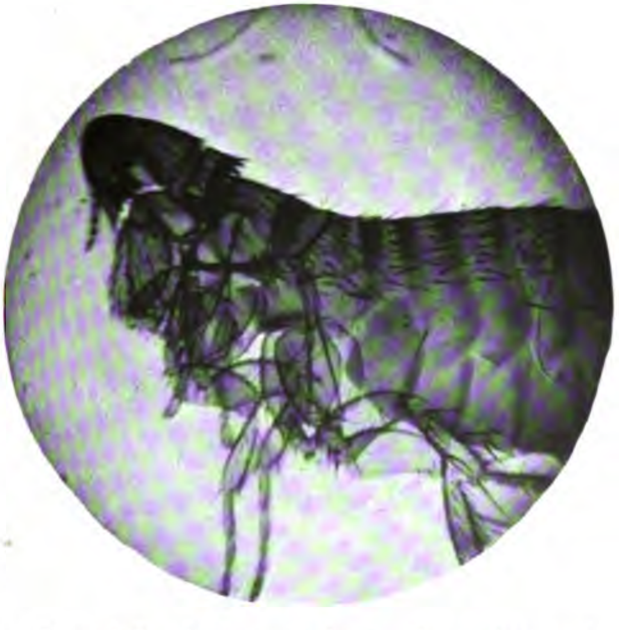
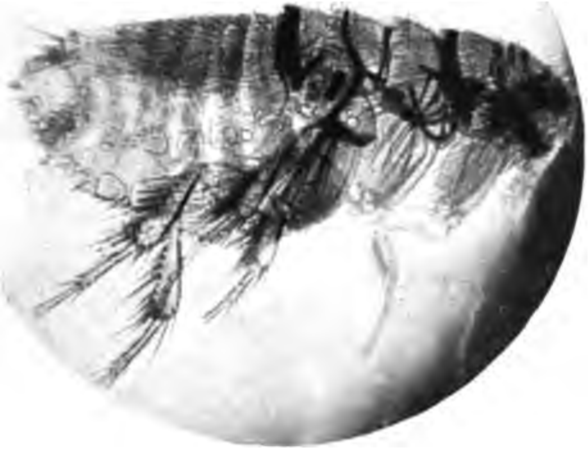


1. *Ceratophyllus fasciatus;* macho. 2. *Ctenopsyllus seguis;* hembra.
(Microfotografías *Cartañá.*)

LAMINA 5

1



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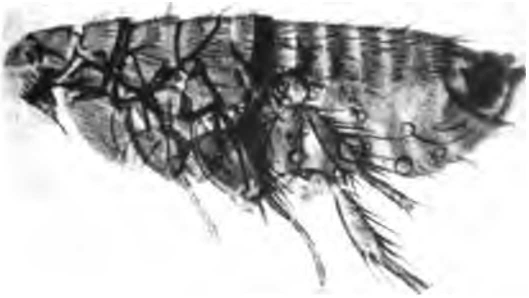


Stenoponia tripectinata.—1, hembra; 2, macho.
(Microfotografías *Cartañá.*)

Género CTENOPSYLLUS KOLENATI.

Sinonimia: *Typhlopsylla* Tasch.
Ctenopsylla Tasch (part.).
Ctenopsylla Wagner.
Ctenophthalmus Oud.
Leptopsylla J. R.

CTENOPSYLLUS SEGNIS SCHÖNCH.

Sinonimia: *Pulex segnis* Schönch.
Pulex musculi Dug.
Ceratophyllus muris Curt.
Ctenophthalmus quadridentatus Kol.
Typhlopsylla mexicana Bak.

Es la pulga del ratón, que no raras veces se halla en la rata. Es también cosmopolita, y en nuestro país se encuentra ampliamente extendida.

Caracteres.—Cabeza angulosa por delante, sin ojos; peine de las mejillas formado de cuatro espinas. Frente con un surco que divide en dos la cabeza, separando la porción occipital del vertex, y tanto una como otra con numerosas cerdas. Protórax con peine de unas 22 espinas. Mesopleura con un engrosamiento mediano vertical. Segmentos abdominales con dos series de cerdas, una de robustas cerdas medianas y otra integrada por cerdas más pequeñas apicales. Espermoteca con cabeza grande y cola fina.

Durante algunas épocas puede encontrarse en un porcentaje bastante considerable en las ratas.

Esta especie puede transmitir la peste en condiciones experimentales, pero en la naturaleza no parece tener importancia en la propagación de aquélla, como lo prueba la rareza de las adenitis cervicales en las ratas, a pesar de la localización típica de la *Ct. segnis* en la región del cuello, como indica Ricardo Jorge y hemos tenido ocasión de comprobar repetidas veces.

Género STENOPONIA JORDÁN Y ROTSCHILD.

STENOPONIA TRIPECTINATA TIRABOSCHI.

Sinonimia: *Histrichopsylla tripectinata* Tiraboschi.

Esta pulga fué descrita por Tiraboschi en 1903 sobre un único ejemplar de macho, proveniente de Italia. Más tarde ha sido citada de Argelia y de las Azores, pero no se ha descrito bien todavía la hembra, por ser indudablemente una especie muy rara. Sobre ella fué descrito el género *Stenoponia* por Jordán y Rotschild en 1911, fundándose en que su receptáculo seminal es único y sus palpos labiales son de dos artejos, mientras los maxilares tienen cinco.

Caracteres.—Es una especie fácilmente diferenciable por la presencia de un peine en el primer segmento abdominal. El peine genal consta de 10 a 12 dientes, y su posición es parecida al de la *Ct. segnis;* carece de ojos, y su cabeza está dividida en dos por el surco antenal. Peine protorácico compuesto de 38 a 40 dientes. El peine del primer segmento abdominal lleva alrededor de 30 púas; los restantes segmentos llevan espinas cortas, cuyo número oscila entre 6 y 10. El macho tiene el dedo móvil del broche largo y de bordes paralelos. La espermoteca de la hembra tiene la cabeza redondeada y la cola aguzada hacia el extremo.

Nuestros ejemplares presentan algunas diferencias con la descripción original de Tiraboschi, que quizá son suficientes para fundar una subespecie. En cambio, coincide con la figura que proporciona Roubaud en *Rongeurs et puces,* por lo que nos abstenemos de darle nombre. Su papel en la transmisión de la peste no se ha comprobado, pero en razón de su rareza no puede llegar a tener importancia epidemiológica.

LA ANATOMIA DE LAS PULGAS

Con objeto de hacer más útil nuestro trabajo, hemos creído conveniente proporcionar una breve descripción, tanto de la morfología externa como de la anatomía interna de las Afanípteros, con lo cual se facilitará la labor de aquellas personas que quisieran dedicarse al estudio de este grupo.

Morfología externa

El cuerpo de una pulga es muy comprimido, con la cabeza ampliamente sentada en el tórax, y todo él presenta un revestimiento de cerdas fuertes dirigidas hacia atrás. Sus patas, como en todos los insectos adaptados al salto, son muy desiguales, y el último par está mucho más desarrollado que los otros dos, mientras sus fémures son gruesos y robustos para albergar a los potentes músculos saltadores.

Cabeza.—Está dividida en dos regiones por un surco mediano inclinado de delante hacia atrás y abajo, en el que están alojadas las antenas, por lo cual recibe el nombre de surco antenal; la anterior es la frente; la posterior, el occipucio. Los ojos son simples y están situados delante del citado surco, como se aprecia en la figura; en muchas especies sufren un proceso de reducción, y en algunas han desaparecido por completo. El borde ventral de la cabeza o mejilla es fuerte y está provisto en algunos géneros de una serie de espinas regularmente dispuestas, que, por su aspecto, recuerdan las púas de un peine, formando el llamado peine genal o de las mejillas.

Las antenas constan de dos artejos basales y una maza apical que representa el tercer artejo, que está formado por una serie de anillos completos en algunas especies e incompletos en otras, cuyo número es bastante variable.

En el reposo están siempre alojadas en el surco antenal, pero el animal puede a voluntad levantarlas o retraerlas. El surco antenal puede estar prolongado hasta el borde superior de la cabeza y constituir una muesca bien marcada, que divide en dos la cabeza y da nombre al suborden de los Fracticipitos, o bien puede concluir antes, o, si llega a alcanzar el borde cefálico, no constituir una escotadura, sino un engrosamiento o *falx*, llamándose Integricipitos los Afanípteros que presentan este carácter.

PIEZAS BUCALES constituídas por:

a) Un labro o labio superior, tapizado en su cara ventral por una prolongación del tegumento bucal o epifaringe, en el que hay numerosas papilas sensoriales. Es largo y está asurcado en toda la longitud de su borde ventral.

b) Dos mandíbulas tan largas como aquél, con sus bordes aserrados en los dos tercios apicales, que son los verdaderos órganos perforadores. En el borde inferior de la cara interna llevan un surco longitudinal en toda su extensión.

c) Dos maxilas que tienen la forma de dos láminas triangulares debajo del borde inferior de la cabeza. Están provistas de dos palpos, que aparecen como dos órganos libres, en la parte anterior, que pudieran ser confundidos con las antenas, de cuatro o cinco artejos.

d) Un labio inferior corto, que se prolonga en dos palpos labiales de cuatro a cinco artejos, cuya cara interna, cóncava, sirve para proteger a las restantes piezas en el reposo.

MECANISMO DE LAS PARTES BUCALES.—Las dos mandíbulas son los instrumentos perforadores, que con un movimiento alternativo de protacción y retracción originan la herida.

Adosándose las mandíbulas a los lados del labro, forman un conducto cerrado, por el que se absorbe la sangre mientras el surco inferior de la cara interna de aquéllas forma un canal cerrado al unirse con el opuesto, por el que se inyecta la saliva, de propiedades irritantes y anticoagulantes.

Las maxilas no toman parte en la perforación de la piel; siempre permanecen a un lado y al otro, ligeramente levantadas.

TÓRAX con los tres segmentos bien aparentes y compuestos cada uno de una pieza dorsal, terguito o noto, una pieza lateral o pleura y una inferior o esternón. La pleura está a su vez compuesta de dos piezas, soldadas en general en el protórax y separadas por una sutura en el mesotórax, así como en el metatórax. La anterior es el episternón y la posterior es el epímero; éste, en el último segmento torácico (metepímero), es muy grande y recubre parcialmente al primer segmento del abdomen. El protórax lleva en ciertos géneros una serie de espinas que constituyen el peine protorácico.

Las patas tienen las caderas muy grandes, articuladas con el esternón de cada segmento torácico; llevan en su extremo un trocánter corto, en el que se inserta el fémur, que es la pieza más fuerte y larga de la pata, en cuyo interior van los fuertes músculos saltadores. En su extremo está articulada la tibia, que, a su vez, lleva en su ápice cinco artejos más, que integran el tarso. El último artejo está provisto por debajo de cerdas llamadas plantares, y en su ápice hay dos robustas uñas.

ABDOMEN.—Tiene diez segmentos, de los cuales los tres últimos están transformados al servicio de la reproducción. Cada uno de ellos va provisto de una o dos series de cerdas, lo cual da origen a la división en familias, siendo en el primer caso basales y en el segundo basales y apicales. El VII terguito abdominal lleva una o varias cerdas largas, llamadas antepigidiales por estar situadas delante del pigidio, que es un órgano de forma de silla de montar y de estructura muy fina.

Cada segmento lleva a los lados un estigma respiratorio.

SEGMENTOS MODIFICADOS.—*Macho:* El VIII esternito es bastante grande y se encuentra en casi todas las especies bastante prolongado, dando al abdomen la forma especial del sexo. El VIII terguito, por el contrario, es corto. El IX esternito tiene una forma muy variable, unas veces cónica, otras mazuda, pero siempre está formado por dos ramas, una ascendente, interna y oculta en el interior del abdomen, y otra posterior, cuyo extremo suele verse cuando el esternito anterior se abre hacia los lados. El IX terguito lleva el pigidio y un par de apéndices, los broches, que tienen dos artejos, el distal, móvilmente articulado sobre el basal, por lo que ha recibido el nombre de dedo móvil del broche. Entre estos últimos segmentos se verifica la salida del órgano intromitente o aedeago, que es muy complicado y siempre con diversas apófisis en forma de bastones quitinosos, cuya variación no suele emplearse en la clasificación. En cambio, el segmento IX, tanto el terguito como los broches y el esternito son extraordinariamente usados en ésta, y conviene distinguirlos bien.

Hembra: En ésta, por el contrario, el terguito VIII es enorme y recubre la porción distal del abdomen. El esternito VIII es pequeño y parcialmente cubierto por aquél. El terguito IX lleva el pigidio como en el otro sexo, además de un pequeño par de apéndices o estiletes; su esternito correspondiente está también, como en el otro sexo, reducido a láminas quitinosas de diversa forma, apareciendo en el extremo del segmento anterior o enteramente oculto en el interior del abdomen. En la hembra se usa mucho en clasificación la forma del VIII esternito y la distribución de las cerdas en el VIII terguito.

Dimorfismo sexual.—Las hembras pueden fácilmente distinguirse de los machos en los Afanípteros por su gran talla, que, aun cuando estén infecundas, es aparente, pero que llega a su máximo cuando están repletas de huevos. También la forma regularmente redondeada del abdomen en la hembra, cuyo borde dorsal y ventral tienen visiblemente la misma curvatura, pueden ayudar a distinguir ambos sexos, puesto que en el macho el borde ventral es redondeado, debido al desarrollo del VIII esternito, pero dorsalmente es casi recto, y en algunas especies incluso ligeramente cóncavo.

Anatomía interna

Desde el punto de vista de la transmisión de la peste, sólo es interesante el estudio del tubo digestivo, que es donde proliferan los bacilos.

Del conducto chupador formado por la unión del labro y las mandíbulas, entra el alimento en la faringe, que es un tubo encorvado, provisto de dos piezas quitinosas, ventral y dorsal, en las cuales se insertan los músculos dilatadores que intervienen en la succión. En algunos ejemplares montados y aclarados se distingue por delante de los ojos, por ser de naturaleza quitinosa. La faringe da paso al esófago, que—como en todos los insectos chupadores, en los que penetran en el interior de su tubo digestivo materias líquidas exclusivamente—es delgado; se extiende hasta el nivel del metatórax, sin presentar ninguna dilatación ni reservorio de alimento.

Al final del esófago se encuentra el proventrículo, órgano más o menos cónico o piriforme, cuyas paredes engrosadas y musculosas han dado origen al nombre de molleja que le dan algunos autores; interiormente va tapizado por un ancho anillo de numerosas espinas dispuestas con regularidad, y cuyo tamaño varía según la zona en que estén enclavadas. Estas espinas funcionan como válvula que impide el retroceso de la sangre al dilatarse la faringe, y, como veremos más adelante, es extraordinariamente interesante en la propagación de la peste.

El estómago, que sigue al proventrículo, es grande y ocupa cuando está distendido buena parte de la cavidad abdominal; su forma es parecida a la de un saco. En su extremo, en la unión con el intestino, recibe el contenido de cuatro tubos de Malpighi, cuya función parece ser semejante a la de nuestros riñones. Detrás de ellos empieza el intestino posterior, que es corto y estrecho, estando su porción terminal o recto engrosado en una ampolla rectal, que lleva seis papilas de pequeño tamaño, indudablemente de función excretora.

I. Anopluros.

La única especie de este orden de insectos que parasita a las ratas de Barcelona es el *Polyplax spinulosus,* que es el Anopluro más común sobre las dos especies españolas de ratas. Pertenece a la familia de los Hematopínidos, y Ewing le incluye en la subfamilia Hoplopleurinos.

POLYPLAX SPINULOSUS BURMEISTER.

Sinonimia: *Pediculus spinulosus* Burm.
Haematopinus spinulosus Denny.
Pediculus denticulatus Nitsch. y Giebel.

Caracteres.—Cabeza redondeada; antenas insertas muy delante, con el primer artejo grande y grueso y el tercero provisto en el macho de un apéndice encorvado característico. Tórax de la forma corriente, en la hembra poco más estrecho que el abdomen. Este en dicho sexo es largo y de bordes casi paralelos, con las pleuras quitinizadas, formando unas placas bien marcadas que dan al abdomen un aspecto aserrado, y llevando una espina a cada lado; del segundo a séptimo terguito, en la hembra, las placas tergales están divididas en dos, y lo mismo sucede con las esternales. En el macho, unas y otras están bien quitinizadas, pero enteras, y el contorno del abdomen no es tan aserrado como el de la hembra, siendo, en cambio, mucho más ancho y corto. Gonópodos de la hembra cortos y pequeños.

Las patas anteriores son marcadamente más pequeñas que las de los otros dos pares y las posteriores son fuertes y robustas.

La larva se parece algo a la hembra en la forma del abdomen, pero es mucho menos peluda.

Su abundancia es mucho mayor durante la época de verano; en cambio, en invierno es difícil encontrar algún ejemplar. Aunque todos los estados se hallan sobre la rata, son las hembras adultas las que se presentan con mayor frecuencia.

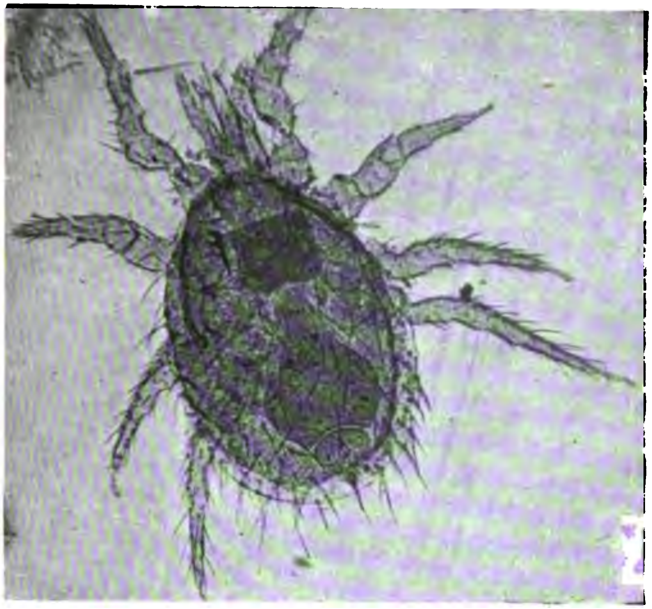
En relación con la propagación de enfermedades, este piojo es el agente transmisor del tabardillo mejicano, ocasionado por una *Rickettsia* que propaga de rata a rata. También transmite la *Bartonella muris* de rata a otra.

III. Acaros.

Tres especies hemos hallado sobre las ratas durante nuestro trabajo, dos de ellos pertenecientes a los Gamásidos y otro a los Sarcóptidos. Los dos géneros de Gamásidos Leláptidos pueden diferenciarse fácilmente con arreglo a la siguiente clave:

LÁMINA 6

1



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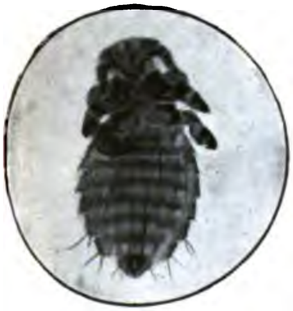

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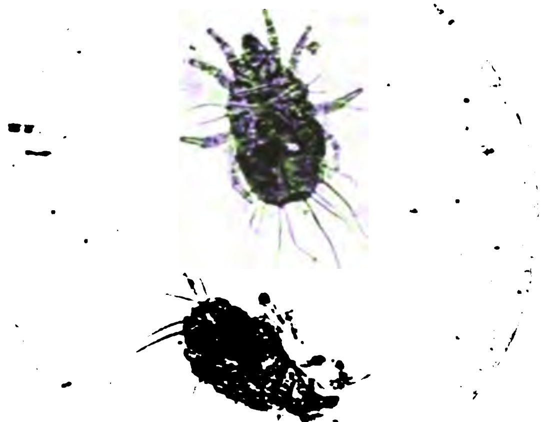


1, *Echinolaelaps echidninus;* 2, *Polyplax spinulosus;* 3, *Tyroglyphus* sp.
(Microfotografías *Cartañá.*)

1. — **Quelíceros en la hembra, con la pinza provista de pequeños dentículos; en el macho, estiliformes. Placa génitoventral muy grande, con su borde posterior cóncavo, por estar en contacto con la placa anal; placas metapodales rudimentarias; escudo dorsal bien desarrollado: ECHINOLAELAPS.**
   — **Quelíceros con la pinza provista de dentículos bien marcados en ambos sexos; placa génitoventral con el borde posterior recto y tocando a la placa anal sólo en un punto; placas metapodales triangulares y grandes: EULAELAPS.**

Género ECHINOLAELAPS EWING.

Sinonimia: *Laelaps* C. L. Koch.

Sólo una especie en las ratas estudiadas.

ECHINOLAELAPS ECHIDNINUS BERLESE

Es el ácaro más frecuente de la rata, y puede decirse que prácticamente es el único que se encuentra. Su localización es preferentemente en la parte posterior del dorso, cerca de la base de la cola, en cuyo sitio suelen encontrarse a veces bastantes ejemplares.

Sólo hemos visto ejemplares hembras, lo que pudiera indicar que tanto los machos como los dos estadios ninfales llevan vida libre.

Caracteres.—Acaros bien quitinizados, con el escudo dorsal de color ocráceo provisto de largas cerdas erizadas; las patas, fuertes y robustas, provistas de ambulacros en todos los pares; la placa esternal de la hembra, de longitud próximamente igual a su anchura; la placa génitoventral, muy ancha en su parte mediana y con el borde posterior marcadamente cóncavo y estrechamente aproximado a la placa anal; ésta tiene una cerda posterior muy larga y dos anteriores más cortas; las placas metapodales rudimentarias.

En relación con la epidemiología de la peste, según experiencias de algunos autores modernos, puede transmitirla en las epizootias de una rata a otra; en cambio, como no parece picar al hombre, no es peligroso para la transmisión de la peste humana.

EULAELAPS STABULARIS C. L. KOCH

Se distingue fácilmente de la especie anterior por su forma más alargada, su coloración más pálida y su menor quitinización.

Caracteres.—Escudo dorsal provisto de numerosos pelos cortos. Placa esternal ligeramente más ancha que larga y con su borde posterior cóncavo; placa génitoventral con numerosas estrías en su parte anterior y muy ensanchada en su parte mediana; su pilosidad es mucho más corta y más densa que en la especie anterior; placas metapodales grandes, mayores que la placa anal y de contorno triangular; la placa anal, con su borde anterior convexo y aproximado en su punto medio a la placa génitoventral, está provista de cinco cerdas cortas. Todo el cuerpo revestido de pilosidad corta, fina y densa.

En relación con el hombre, esta especie ha sido señalada parasitizando a algunos soldados, a los que producía molestias muy considerables con sus picaduras.

Es interesante este hecho, puesto que, en el caso de que la especie de que nos ocupamos pudiera infectarse con el virus pestoso, podría en algunas circunstancias inocularlo al hombre.

FAMILIA SARCOPTIDOS

Género NOTOEDRES RAILLIET

Existen dos especies de este género, que parasitizan a las ratas, si bien una de ellas se presenta más especialmente en la rata negra y la otra en la rata gris. Nosotros no hemos hallado más que una.

NOTOEDRES MURIS MEGNIN

CARACTERES.—Con chupadores en el primero, segundo y cuarto par de patas en el macho y en los dos primeros en la hembra; abertura anal dorsal.

Los pelos del dorso en la hembra son fuertes y parecen pequeños bastoncitos.

Este parásito se encuentra localizado de preferencia en las orejas y en el hocico; aquéllas se recubren de unas excrecencias córneas que le dan el aspecto de lóbulos, debajo de las cuales abundan ejemplares de todos los estadios. Nosotros únicamente lo hemos observado durante los meses de invierno, si bien debe abundar en toda época.

CAPÍTULO III

ESTUDIO BIOLOGICO DE LAS PULGAS DE LAS RATAS

I.—La captura de ratas y la recolección de sus ectoparásitos

Para nuestro estudio hemos utilizado las ratas capturadas por la brigada de desratización del Instituto Municipal de Higiene, cuyo centro tiene perfectamente organizado este servicio, que se verifica de manera sistemática, por medio de rateras del modelo corriente, en forma de nansa, con un embudo de entrada, que en su parte inferior tiene una trampa en balancín, por la que el animal cae dentro de ella.

El digno director de aquel Centro puso a nuestra disposición el material proveniente de esta desratización, y gracias a él hemos podido economizar tiempo y medios, que de otra manera hubieran sido indispensables.

Las brigadas cazarratas colocaban las rateras por la tarde, y a la mañana siguiente recogían las que contenían ratas, que eran transportadas enseguida al laboratorio.

El procedimiento empleado para la captura de los ectoparásitos consistía al principio en separar las ratas capturadas en distintos sacos, que se sometían a la acción del gas cianhídrico, para matarlas. Los sacos se llevaban después al laboratorio, en donde se sacaban las ratas y se peinaban con ayuda de unas agujas, para buscar aquellos parásitos que estuvieran aún sobre el huésped, registrando después los sacos para recoger los ejemplares que en ellos quedaran.

Más tarde modificamos el procedimiento, sometiendo las rateras íntegras a la acción del gas cianhídrico, con lo cual se evitaba el tener que aislar las ratas, siempre molesto y que hacía perder bastante tiempo en esta manipulación. El único inconveniente de este método estriba en que algunas pulgas, por ser más resistentes a la acción del gas que las ratas, pudieran escapar y permanecer en la cámara de cianhidración; pero esto se obviaba con aumentar un poco la dosis de gas.

De este modo observamos que la cifra total difería muy poco en ambos procedimientos. Presenta también esta marcha la ventaja de que si alguna rata hubiera sido pestosa, el riesgo de que las pulgas infectadas la abandonasen se reduciría al mínimo.

Los ejemplares, como en el sistema anterior, eran peinados cuidadosamente para recoger todos los ectoparásitos.

Captura de pulgas vivas

Con objeto de obtener pulgas vivas para los experimentos de cría y de infección experimental, nos valimos de otro medio. Las rateras, y si éstas eran grandes las ratas aisladas, cogidas con ayuda de unas pinzas fuertes de presión, se sumergían rápidamente en la pila del agua, que era de mosaico blanco; parte de las pulgas abandonaban al huésped y se recogían en la superficie del agua, y la otra parte que quedaba sobre él se capturaban al peinarlas mientras estaban húmedas, con lo que se impedía que pudieran saltar.

Este sistema es mejor que el empleado por uno de nosotros (J. G. C.) en 1925 para hacer la primera encuesta sobre las pulgas de las ratas del puerto de Barcelona, que consistía en estrangular a las ratas con un trozo de cuerda e introducirlas después en un saco blanco, en donde quedaban las pulgas vivas, pero con el riesgo de que podían escapar fácilmente.

De todas maneras, es menester tener gran cuidado en esta manipulación si se quiere conseguir que no se escape ningún parásito, y lo mejor es trabajar sobre un cristalizador lleno de agua o sobre una mesa cubierta de un trapo blanco empapado de agua, en el cual las pulgas que saltan quedan adheridas; este método seguido por nosotros en nuestro trabajo nos ha dado resultados excelentes.

II.—Captura de pulgas sobre animales colocados como cebo

A continuación exponemos un caso en el que empleamos el cobaya como cebo para recoger pulgas de una casa en la que se había presentado un caso sospechoso de peste.

En el sitio de captura de la rata núm. 1.189 (calle de Alibey, números 162 a 172) hubo un caso sospechoso de peste, que, aunque luego resultó bacteriológicamente negativo, atrajo nuestra atención, desde el momento de la denuncia, hacia el sitio indicado. Se trata de un establecimiento formado por varios almacenes con fachada a la calle de Alibey, esquina a Castillejos, y con un pasaje intermedio. Pertenece a un trapero mayorista (drapaire) en el sentido que en Barcelona se da a esta palabra, o sea, que se dedica no sólo a trapos viejos, sino a todo lo que sea material inservible, tal como desechos de maquinaria, papeles, hierros, latas, tubería, alpargatas, neumáticos viejos, etc. El trabajo de la sección de trapería propiamente dicha consiste en la compra al pequeño trapero de su recogida diaria, y luego se practica allí mismo la selección y prensado con prensa hidráulica para formar las balas de trapo.

Uno de los trabajadores empleados en esta sección fué enviado al hospital de infecciosos como sospechoso de peste, por presentar una adenitis inguinal. Visitamos la habitación en que vivía, en un barrio del extremo opuesto de donde trabajaba, y como no ofreciera nada de particular desde nuestro punto de vista, dirigimos nuestra encuesta hacia el local de la calle de Alibey, disponiendo la colocación de rateras.

La cantidad de pulgas existentes en el local de los trapos era tal, que después de la primera visita pudimos capturar sobre nuestro mozo de laboratorio, que fué intensamente picado, 25 pulgas vivas, pertenecientes a las especies siguientes: 17 *Pulex irritans* y ocho *Ct. felis*.

Aunque este individuo estaba vacunado, procedimos rápidamente a la inoculación por vía intraperitoneal al cobaya del producto del triturado y emulsión de dichas pulgas, con intención de inyectarle suero antipestoso, caso de que dicha inoculación no hubiese resultado negativa. Aquel mismo día dejamos siete cobayas, cada uno en una jaula ratera de alambre, y repartidos por todo el local de la trapería con el doble objeto de capturar pulgas para su estudio y de ver si se infectaba algún cobaya espontáneamente. A los siete días fueron sacrificados tres de los cavias, encontrándoseles siete, seis y una pulgas, respectivamente.

Las siete primeras eran: cinco *X. cheopis*, una *P. irritans* y una *Ct. felis*; las del segundo cobaya, dos *X. cheopis*, una *P. irritans*, dos *Ct. felis* y una *Cer. fasciatus*. En el tercer cobaya se recogió una *X. cheopis*.

La inoculación del triturado de estas pulgas tampoco dió resultado positivo.

Los otros cuatro cavias fueron separados y puestos diez días en observación en el laboratorio, sin que ninguno de ellos muriese ni ofreciera particularidad alguna.

En resumen, pues, las tres pruebas practicadas fueron completamente negativas.

El hecho de que se encontraran tan pocas pulgas en los cobayas indicados, cuando tantísimas había el primer día que fuimos, queda explicado por la desinfectación intensa que los propietarios de la trapería efectuaron en el local desde el primer día para darnos la sensación de una limpieza que, en realidad, no habían observado hasta entonces.

III.—Cría de pulgas

En diferentes ocasiones hemos ensayado el cultivo de pulgas en el laboratorio, con el doble objeto de estudiar algunos puntos de su biología y de poseer abundante material para las infecciones experimentales, ya de peste, ya de otros virus análogos, por los insectos adultos.

Aprovechando una de nuestras estancias en Barcelona, en el mes de agosto empezamos ya dicho cultivo, valiéndonos de un sistema parecido al que Roubaud preconiza para la cría de flebotomos.

Consiste en hacer un bloque de escayola, del tamaño de una cápsula de Petri, en cuya cara superior, estando aún blanda la masa, se coloca un vidrio de reloj con objeto de producir una pequeña oquedad, que ha de servir para colocar posteriormente el medio de cultivo.

En esa pequeña concavidad se coloca una capa de arena fina, que se espolvorea con la sustancia nutritiva, en este caso, hígado pulverizado. Para conservar la humedad se tapa ese hueco con un embudo invertido, de tamaño apropiado, que se cubre en sus bordes con arena húmeda, y cuyo tubo superior se tapa con un trozo de algodón que impide la excesiva evaporación.

El bloque de escayola se coloca dentro de una cápsula de Petri llena de agua, o bien sobre arena bien mojada, para que vaya penetrando la humedad y se mantenga siempre considerable en el medio nutritivo.

Una vez recogidas las pulgas vivas, y determinadas la de cada especie, se eligen los ejemplares de hembras que estén grávidas, para asegurar el éxito del cultivo, y se colocan en cada uno de estos bloques dos o tres ejemplares de una misma especie.

Al cabo de unos días, las pulgas han realizado la puesta, y los huevos colocados en la superficie de la arena empiezan a dar origen a las larvas. Todos estos cultivos se perdieron sin dar lugar a adultos.

En el mes de noviembre volvimos a reanudar los cultivos, empleando, además de este sistema, el método ideado por Leeson sobre ratones vivos.

Por el sistema de cría en moldes de escayola, no pudimos tampoco obtener el desarrollo completo, debido a los hongos que crecen sobre la sustancia nutritiva y llegan a cubrir por completo toda la excavación, impidiendo el desarrollo de las larvas, por lo cual, en el caso de querer seguir este sistema, ha de emplearse arena e hígado completamente esterilizado.

Cría sobre ratón por el método Leeson.

En un local de 30 centímetros de alto y de 7,50 de diámetro se dispone una capa de arena fina, en el fondo, de seis milímetros de altura. Sobre ella se esparce una pulgarada de hígado de vaca seco, pulverizado, de manera que quede bien extendido.

Para proporcionar agua al ratón, recomienda Leeson un dispositivo que consta de un tubo de 2,5 cm. de diámetro y de 7,5 cm. de longitud, cerrado en sus extremos con dos tapones de corcho, de los cuales el que ha de quedar en la parte inferior se perfora para permitir el paso de un tubo más estrecho, de 0,5 cm. de diámetro y de tamaño conveniente para que sobresalga por debajo del tapón unos 3,5 cm. El extremo de este tubo se lleva a la lámpara hasta que su orificio se reduzca próximamente a la mitad, y por medio de un alambre se sujeta, y haciéndole un asa, como se ve en la figura, se mantiene en la boca del frasco.

El ratón puede lamer el extremo inferior del tubo, sacando bastante agua para su mantenimiento, sin que haya peligro de que aquélla caiga gota a gota, una vez bien tapado el corcho superior. Sin embargo, conviene graduar bien el orificio del extremo y convencerse de que el agua no gotea, pues, en caso contrario, todo el fondo se llena de agua y las larvas de pulgas no pueden sobrevivir.

Para alimentar el ratón se emplea la avena previamente esterilizada, pues, de lo contrario, como advierte el autor y como a nosotros nos ha ocurrido, se desarrollan en ella una serie de ácaros que acaban con el cultivo.

Cuando todo se ha dispuesto y se ha colocado, la cucharada pequeña de avena para la manutención del ratón, se tapa el frasco con un tapón metálico agujereado, sobre el que se coloca un trozo de tela de muselina para cubrirlo bien. Con el primero se evita que los ratones puedan escapar (cosa muy fácil, sobre todo, en el caso de algunos ratones muy ágiles), y el segundo sirve a su vez para impedir que las pulgas puedan salir a través de los orificios del tapón metálico.

Leeson recomienda para la cría de *Ceratophyllus* mantener estos frascos a la temperatura de la habitación, y para la *Xenopsylla cheopis*, llevarlos a la estufa a 23°.

Inconvenientes observados con esta técnica.

En los primeros cultivos observamos que el ratón, sobre todo cuando se coloca sobre él la pulga del perro, *Ctenocephalides canis*, u otra especie, que, en general, no parasitice a los roedores de ese género, se torna inquieto por la viva molestia que le ocasionan sus picaduras y acaba casi siempre por devorar todos los ejemplares.

Para obviar este inconveniente recurrimos a cortar los incisivos a los ratones, alimentándolos con cebada muy cocida y empapada de agua, bastando una ración cotidiana de doce granos para su mantenimiento.

El método de Langeron para la cría de pulgas, ensayado a su vez en

Madrid, nos resultaba demasiado sucio y no pudimos obtener buenos resultados, quizá por la mala dosificación de la comida. Estriba en cortar los incisivos a un ratón o a una rata y colocar sobre ella, en un frasco, las pulgas de la especie que se quiere criar. Para alimentarlas se emplea pan empapado en leche; pero presenta el inconveniente de que se ensucia todo el fondo del frasco, terminando por quedar empapada toda la arena que se ha colocado en él con los excretas líquidos de la rata y la leche de las migas de pan, que a su vez fermenta y produce un olor insoportable.

Cultivos realizados.

a) Sobre el bloque de escayola.

En agosto emprendimos seis cultivos con *Xenopsylla cheopis, Ceratophyllus fasciatus, Ctenopsyllus segnis* y *Ctenocephalides felis*. De la primera se emprendieron tres. Como hemos advertido anteriormente, estos cultivos se perdieron, y sólo uno de ellos llegó a producir larvas próximas a la pupación, que murieron sin verificarla.

En algunos de ellos, las pulgas morían antes de hacer la puesta adheridas a las paredes del embudo, que se empañaban por la humedad interior.

En noviembre emprendimos otra serie de cultivos con cada una de las seis especies más frecuentes de la rata; pero todas ellas murieron a consecuencia del enmohecimiento del medio nutritivo.

b) Por el método de Leeson.

Primera serie: Se preparan cuatro bocales, con los siguientes ejemplares de pulgas:

1.° 40 *Xenopsylla cheopis.*
2.° 68 *Ctenopsyllus segnis.*
3.° 2 *Ceratophyllus fasciatus.*
4.° 24 *Ctenocephalides felis.*

Todos los cultivos mueren por temperatura demasiado baja. Puede notarse la predilección del *Ctenopsyllus segnis* por la región cervical, como indica Ricardo Jorge.

Segunda serie: Se disponen 15 bocales con las siguientes especies:

Números	1-3.	*Xenopsylla cheopis.*
—	4.	*Ctenocephalides felis.*
—	5-11 y 15.	*Ceratophyllus fasciatus.*
—	12-14.	*Ctenopsyllus segnis.*

En esta serie murieron todos los ejemplares por la temperatura baja, y al mismo tiempo, por la diferente temperatura del interior del frasco y de la habitación, que hace que se condense el vapor de agua en el interior de aquéllos, con lo cual las pulgas se quedan en su mayor parte adheridas a las paredes, muriendo sin lograr poner los huevos.

Tercera serie: Emprendemos los cultivos en dos bocales, con las pulgas de dos especies, ya que no nos fué posible encontrar otras durante aquellos días. Se modifica el cultivo en el sentido señalado anteriormente, cortando los incisivos a los ratones, aumentando la capa de arena para que pueda absorber mejor los excretas, la tapa metálica se sustituye con un tela metálica para que la humedad no sea muy diferente de la existente en el ambiente externo, al objeto de que no se condense el vapor de agua en las paredes.

Colocamos los frascos dentro de una vitrina calentada por un mechero de Bunsen para mantener aproximadamente una temperatura de 18 a 20°.

Cultivo número 1. *Ctenocephalides felis.*
— — 2. *Ceratophyllus fasciatus.*

A los dos días de colocados estos cultivos muere uno de los dos ratones, que se reemplaza inmediatamente por otro; éste a su vez, a los cuatro días, muere, y es sustituído por un tercer ratón, y éste, a los seis, por otro.

Como resultado de estos cultivos, en el número 2 se recoge una ninfa, muriendo en cambio todas las larvas en el número 1.

Cuarta serie: En la sección de entomología de la *London School of Hygiene*, uno de nosotros (P. C. C.), pensionado por la Comisión de Investigaciones Sanitarias, experimentó con el Dr. Leeson la técnica de cría de pulgas ideada por éste, pudiendo observar el extraordinario cuidado que hay que tener para evitar que se invadan los cultivos por una especie de ácaro del género *Tiroglyphus*, parásito de los cereales, y que se introduce en las crías con la avena destinada a alimentar al ratón. Esto obliga a esterilizar los granos antes de ponerlos en los frascos y a tener cuidado de no pasar nunca de un cultivo a otro sin haber efectuado previamente esta esterilización.

Los frascos destinados a este objeto se suelen tener en estufas, a temperatura constante, sin lo cual la probabilidad de un fracaso es mucho mayor. El número de pulgas que se coloca sobre el ratón es muy considerable, para compensar la pérdida de las que son devoradas por el animal.

Aparte de estos cultivos en serie, realizamos, bajo la dirección del citado especialista, la técnica de cultivos aislados, para comprobar los efectos de los distintos factores en el desarrollo de las pulgas, con lo cual está realizando el referido entomólogo inglés unas observaciones sumamente interesantes y de verdadera importancia desde el punto de vista de la epidemiología de la peste, que consisten en el estudio de la influencia que ejerce sobre el desarrollo de la larva la diferente alimentación. Para ello hace cultivos separados, disponiendo varias series de frasquitos conteniendo un huevo cada uno, de una especie determinada, y en cada una de estas series varía la sustancia alimenticia destinada a la larva que saldrá del huevo, distribuyendo en una polvo de sangre desecada; en otra. trocitos secos de trapo empapados en sangre, salvado, polvo de hígado, etcétera.

Haciendo así un gran número de ensayos para cada alimento y en distintas especies de pulgas, estudia para cada sustancia el número de huevos que han dado lugar a eclosión de larva, el número de larvas que han llegado a pupa y a insecto adulto y el tiempo que ha tardado esta evolución en conjunto y en cada una de sus fases, observando y deduciendo las diferencias que van del empleo de una substancia alimenitcia a otra. Hemos seguido estas experiencias, durante nuestra estancia en Londres, con el Dr. Leeson; pero no nos extendemos más sobre estas técnicas ni sobre sus resultados, que serán publicados en breve por su autor.

Estriba este otro método en recoger los ejemplares de hembras grávidas y depositarlas aisladamente en tubos de unos dos centímetros de diámetro por cuatro de longitud, en cuyo interior se deposita un papel negro. Una vez que la puesta se ha efectuado (a las veinticuatro o cuarenta y ocho horas), se cortan trocitos del papel que lleven un huevo y se colocan en tubos iguales a los descritos con un poco de arena fina en su fondo y con una escasa cantidad de uno de los alimentos citados en su superficie. Estos tubos se colocan en unos aparatos destinados a controlar la humedad por el método de las distintas mezclas de ácido sulfúrico y agua, que después de cerrados herméticamente mantienen una humedad relativa constante, manteniéndose en estufas a temperaturas determinadas. En el esquema de la figura 1, tomado de Leeson, se expresan las cantidades necesarias para mantener la humedad relativa que se desee.

Por el método del cultivo seriado, al regreso de Inglaterra, llegamos a obtener, durante el verano de 1933, tres generaciones consecutivas de *C. fasciatus* y de *X. cheopis,* desarrollándose mucho más fácilmente el primero a pesar de la elevada temperatura que alcanzaban los frascos en la época de los cultivos cuando la temperatura del ambiente a la sombra marcaba los 33°. El tiempo de desarrollo en esta época invertía aproximadamente un mes y medio.

Esto demuestra que, en la práctica, el método Leeson da excelentes resultados para la cría de pulgas en serie, y solamente la falta de algunos requisitos contribuía a nuestros fracasos primeros.

Uno de ellos, y no de los menos importantes, era el realizar los cultivos a la temperatura excesivamente baja a que llegaba el laboratorio durante los meses de invierno, que originaba muchas veces la muerte tanto de los ratones como de las pulgas.

Hemos de hacer resaltar que cada uno de estos ensayos infructuosos implicaba una pérdida considerable de tiempo, y que transcurrían bastantes días en muchas ocasiones, hasta encontrar ejemplares de pulgas en número suficiente para emprender de nuevo los cultivos.

Los ataques del *Tyroglyphus* a nuestros cultivos nos hizo pensar en la posibilidad de utilizarlo como parásito auxiliar en la lucha contra las larvas de pulga. En efecto, este ácaro acaba casi siempre por ocasionar la muerte de la larva, y en las contadas veces en que ésta llega a tejer el capullo se encierra en su interior y termina por matar a la ninfa antes de que se produzca la eclosión del adulto.

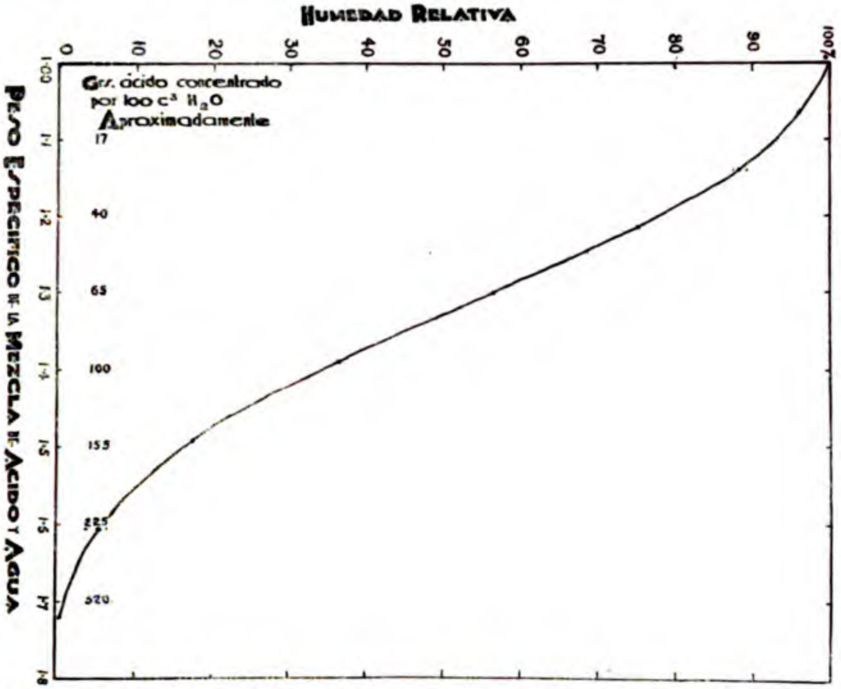


Figura 1.—Porcentajes de ácido sulfúrico y agua para obtener grados de humedad determinada, según Leeson.

Las razones de su posible empleo como parásito auxiliar en esa lucha son, por una parte, su eficacia para matar las larvas, y por otra, el ser inofensivo para el hombre.

Sin embargo, recientes investigaciones de autores italianos han descubierto su intervención en la propagación de la peste, y por lo tanto, a pesar de que sea poco probable la infección por sus picaduras, obliga a no realizar cultivos del ácaro con dicho objeto sin investigar antes a fondo su papel en esta enfermedad.

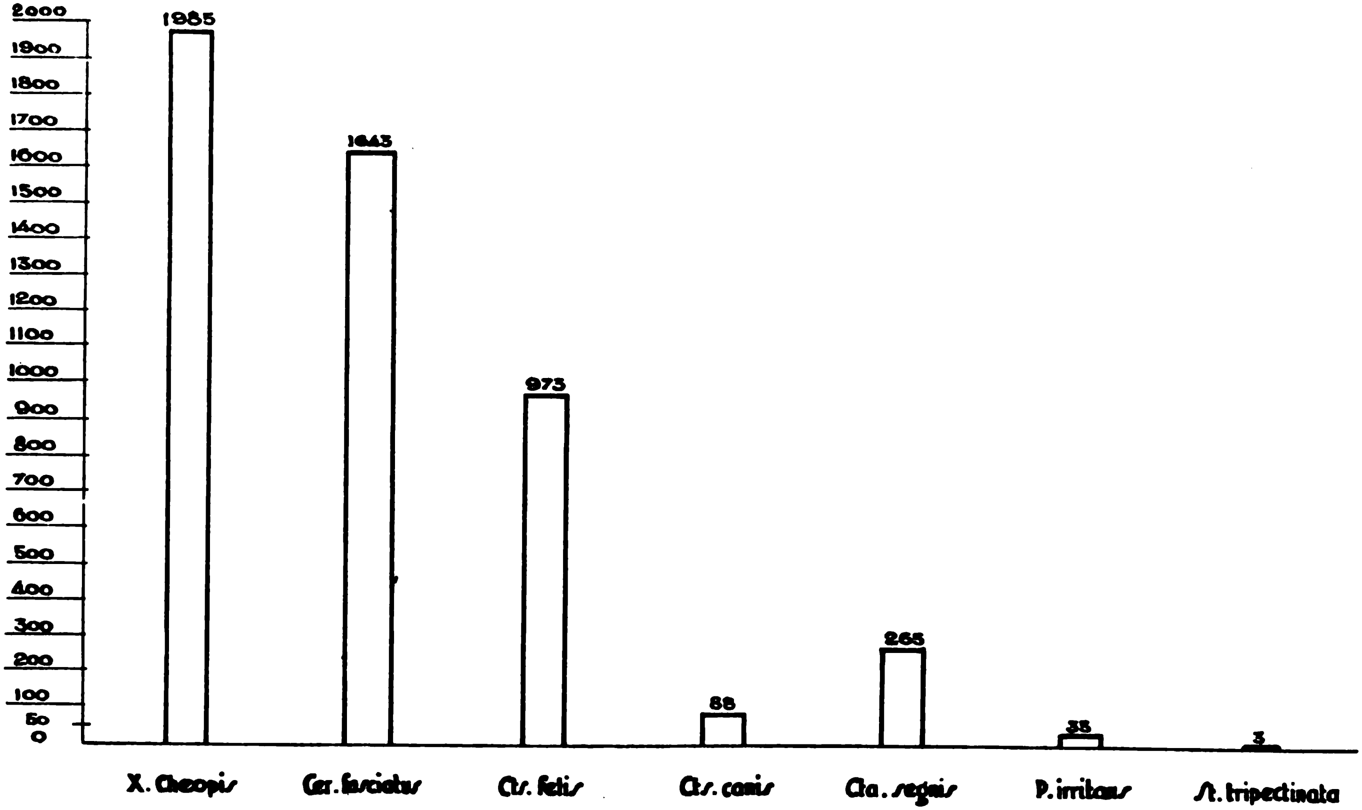


Fig. 2.—Número total de pulgas examinadas, distribuídas por especies.

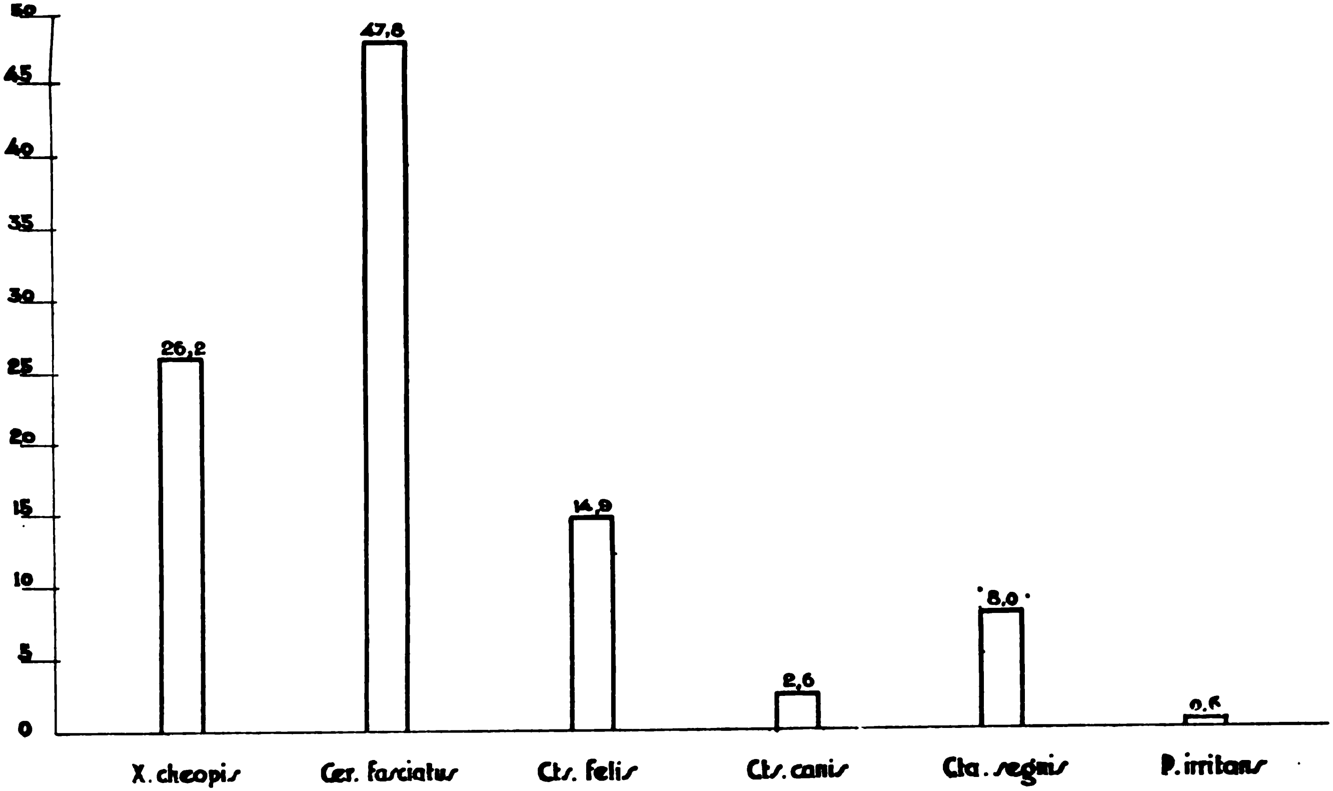


Fig. 3.—Porcentaje medio anual de las cinco especies de afámpteros más frecuentemente encontradas.

del año 1.565 ejemplares con un total de un 47,8 por 100 a su favor, es decir, casi la mitad de las pulgas recogidas.

La especie que le sigue en importancia es la *X. cheopis,* que alcanza la cifra de 857 ejemplares con un 26,2 por 100. En cuanto a las demás encontradas son, por orden numérico: *Ct. felis,* con 488 ejemplares, o sea un 14,9 por 100; la *Ct. segnis,* con 262 y un 8,0 por 100; el *Ct. canis,* del cual se han recogido 87, o sea un 2,6 por 100; el *P. irritans,* del que se han recogido nueve ejemplares, lo que arroja un 0,3 por 100, y por último, la *St. tripectinata,* con dos ejemplares, o sea que no llega al 0,1 por 100. (Véase los gráficos de las figuras 2 y 3.)

Estos datos, tomados de los recogidos durante los doce meses de marzo de 1932 a febrero de 1933, hacen resaltar a la vista el escaso número de pulgas en relación con las ratas; en efecto, aun contando todas las especies, apenas alcanza un índice total anual de 0,6, es decir, un número prácticamente muy bajo.

Según las investigaciones del Dr. Taliaferro Clark, cuando el número medio anual de pulgas por rata es inferior a cinco y la pulga predominante no es *X. cheopis,* la colectividad puede ser considerada como relativamente no infectable; cuando es superior a cinco esta media y la pulga predominante no es de esa especie, la colectividad es infectable y pueden surgir pequeñas epidemias, y cuando la media anual es superior a cinco y la pulga predominante es *X. cheopis,* sería la colectividad altamente infectable.

Aunque esta media sea un tanto aleatoria, creemos que puede proporcionar una orientación general suficiente.

Barcelona entraría en el primer grupo, es decir, entre las poblaciones relativamente poco infectables.

Con ello concuerda la escasa importancia que alcanzan los brotes epidémicos de peste que en los recientes años se han presentado en Barcelona, y que han sido prontamente yugulados, en parte, gracias a la actividad desplegada por los servicios sanitarios para su extinción; pero también ayudada por el escaso número de ectoparásitos que viven sobre las ratas de la población.

Naturalmente, el número de pulgas es muy variable, según los distintos meses, y su índice es muy diferente en cada rata.

Refiriéndose a la *X. cheopis,* el mes que se ha presentado en mayor proporción ha sido en julio, en cuya fecha, sobre 448 ratas se han encontrado 290 ejemplares de esa especie, o sea un índice de 0,7 por rata. En cambio, el índice menor lo ha dado el mes de febrero de 1933 con 844 ratas y 17 pulgas de esa especie, o sea un índice de 0,02 por 100.

Aun en la época de más abundancia, el porcentaje no resulta favorable para la propagación de las epidemias pestosas.

En cuanto al *C. fasciatus,* su número índice se acerca mucho a uno durante los meses de febrero, abril, mayo y junio de 1932, siendo su mínimo de 11 ejemplares sobre 344 ratas, es decir, un 0,03 de índice en el mes de octubre.

Respecto a las restantes especies, sólo el *Ct. felis* llega a tener una cifra máxima en julio de 274 ejemplares sobre 448 ratas capturadas, con un índice de 0,6, siendo las otras especies bastante menos numerosas.

Si nos referimos a los porcentajes relativos de las distintas especies, se presenta el hecho bien marcado de la curva francamente opuesta de la *X. cheopis* y el *Ceratophyllus fasciatus.* (Véase el gráfico de la figura 4.)

La curva de la *X. cheopis* sigue una marcha paralela, pero retardada en dos meses con respecto a la de la temperatura, alcanzando su máximo en los meses de septiembre y octubre, en los cuales predomina claramente sobre las restantes especies. A partir de octubre hay un brusco descenso de su porcentaje, que en el mes siguiente es sobrepasado por la de *C. fasciatus,* y continúa su marcha descendente hasta el mes de febrero, dando el mínimo en el de enero.

El retraso con relación a la temperatura es seguramente debido al tiempo de desarrollo larvario.

En cuanto a la relación posible de su curva con la de la humedad, parece estar directamente relacionada con ella en los meses fríos; pero en agosto y septiembre, en cuyos meses la humedad relativa desciende el número de las *Xenopsylla,* sigue su marcha ascendente. (Fig. 4.)

El *Ceratophyllus fasciatus* muestra al comienzo del estudio, es decir, en el mes de marzo, un porcentaje elevado, que sigue manteniéndose muy alto hasta llegar al mes de mayo, en el que se inicia un descenso, que desde el mes de junio es muy rápido, y llega a su cifra mínima en el mes de septiembre, para empezar de nuevo un trayecto ascendente, que culmina en los meses de enero y febrero últimos. Su relación inversa con la curva de la temperatura es tan marcada, que cualquier oscilación de aquélla se muestra en proporción inversa en el porcentaje de esta especie. En cambio, con la humedad no parece mostrar una relación tan evidente, si bien su curva es en general también inversamente proporcional a ella.

Dada la temperatura de Barcelona, estos datos están perfectamente de acuerdo con nuestros conocimientos acerca de la biología de estos dos insectos, o sea que la *X. cheopis* o pulga tropical, importada, tiene una temperatura óptima de desarrollo de 28° y humedad de 75-80 por 100, mientras que *C. fasciatus* requiere una humedad de 70°, y su temperatura óptima es 18°. Al exponer los brotes epidémicos de peste en Barcelona volveremos a hablar de estos datos y de su relación con la epidemiología de la citada enfermedad.

En cuanto al *Ct. felis,* está relacionado directamente con la temperatura, como la *X. cheopis,* alcanzando su porcentaje máximo en agosto, mien-

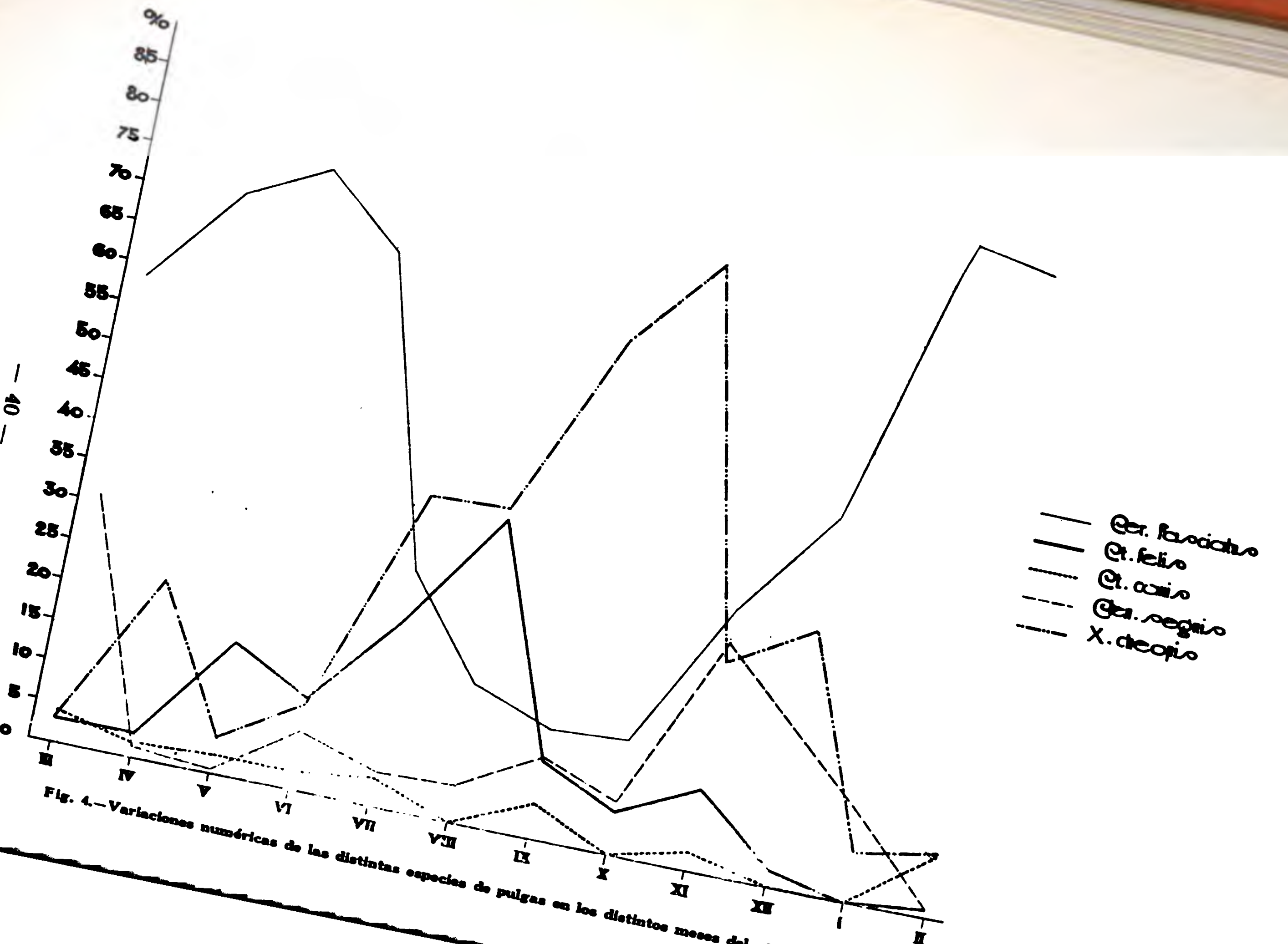


Fig. 4.—Variaciones numéricas de las distintas especies de pulgas en los distintos meses del año, expresada

tras su mínimo número se presenta en los meses de enero y febrero. En agosto llega casi a igualar numéricamente a esta última especie.

La *Ct. segnis* presenta un máximo en marzo y otro en noviembre, casi de igual importancia, y durante los meses restantes se presenta en un número relativamente pequeño, que sufre ligeras variaciones no muy importantes, quizá por necesitar temperaturas moderadas para su desarrollo, no excesivamente calientes o frías.

Estas cuatro especies son indudablemente las más interesantes, por tener un porcentaje relativamente elevado, al menos durante algunos meses. En cambio, *Ct. canis* no pasa nunca de un 5 por 100, sin oscilaciones de importancia, inclinándonos a creer que los altos y bajos de su curva se deben más bien a circunstancias ocasionales que a factores climáticos.

La *P. irritans* se encuentra tan pocas veces sobre las ratas, que parece indicar que ésta no constituye un huésped deseable para ella, y por tanto, sólo por excepción se dirige a dichos roedores a falta de otro huésped más adecuado. Su número relativo es siempre tan escaso, que no ha podido ser representada en la gráfica, en la cual no aparecen más que las cinco especies que anteceden.

Por último, la *St. tripectinata* es una especie esencialmente rara, y no hemos logrado encontrar más que dos ejemplares durante la época fría del año. Aunque parece ser especie propia de las ratas, no tendría nada de extraño que su huésped habitual fuera otro animal poco abundante, por lo que no se ha logrado capturar ningún ejemplar sobre él. De todas las consideraciones que anteceden se pueden deducir las siguientes

Conclusiones.

1.ª La curva de la *X. cheopis* está en relación directa con la temperatura; pero con un retraso aproximado de dos meses, quizá debido al desarrollo larvario.

2.ª El porcentaje máximo anual de *X. cheopis* es en el mes de octubre, y su mínimo, en el mes de enero.

3.ª La relación entre la humedad y la curva de esta especie no muestra una correspondencia estrecha, si bien tanto por alcanzar su cifra mínima 63 por 100 como por ser en general cercana a los 70 por 100, permite el desarrollo de la larva y se aproxima a la cifra óptima.

4.ª El *C. fasciatus,* al contrario de la especie anterior, está en relación inversa de la temperatura, siendo su máximo en enero y febrero, y su mínimo, en septiembre y octubre.

5.ª En cuanto a la humedad, y quizá por las razones que acabamos de indicar, no muestra una relación estrecha.

6.ª La curva del *Ct. felis,* como la de la *X. cheopis,* es inversamente proporcional a la temperatura, alcanzando su máximo en agosto, cuando la temperatura es mayor, y descendiendo inmediatamente después, conservándose baja todo el resto del año.

7.ª La curva del *Ct. segnis* tiene dos vértices máximos en marzo y en noviembre, siendo mucho más bajo su porcentaje en los restantes meses del verano; parece necesitar temperaturas moderadas.

8.ª *Ct. canis* conserva un porcentaje bajo y con ligeras oscilaciones durante todo el año.

9.ª La rata no parece ser huésped apropiado para la *P. irritans*, que sólo acude a ella excepcionalmente, quizá por carencia de otro animal más favorable.

10. La *St. tripectinata* es en Barcelona, como en los restantes lugares en que se ha hallado una pulga rara, que hasta ahora no se ha encontrado más que en los primeros meses del año, durante el invierno.

11. Como conclusión general, que se desprende del escaso número de ectoparásitos por rata, y de que la *X. cheopis* no es la especie predominante en el año, Barcelona entra en el grupo de las poblaciones relativamente no infectables, en la que los brotes epidémicos son fácilmente dominados.

CAPITULO V

LA ESTRUCTURA DEL PROVENTRICULO Y EL MECANISMO DE LA TRANSMISION DE LA PESTE POR LAS PULGAS

Los autores que han contribuído de una manera más intensa al conocimiento de la manera de transmitir las pulgas el bacilo pestoso han sido Bacot y Martin en el año de 1914.

Cuando se alimentan sobre animales pestosos las pulgas de las dos especies estudiadas por Bacot, *X. cheopis* y *Cer. fasciatus*, al cabo de un día o dos aparecen en su proventrículo unas motas, que van haciéndose cada vez mayores, debidas a la aglomeración de bacilos pestosos. Siguiendo su desarrollo, la entrada del estómago va poco a poco obstruyéndose, y al cabo de cierto tiempo, debido a su proliferación en el proventrículo, se produce un bloque de bacilos lo suficientemente fuerte para obstruirle por completo. A este fin coadyuva la forma de éste y las espinas de que está provisto, según puede verse en el capítulo correspondiente.

La pulga en este estado recibe el nombre de pulga bloqueada, y aunque su faringe, por la acción de los músculos dilatadores, puede aspirar sangre, ésta no puede pasar el estómago, sino que al contraerse aquélla llega a ser regurgitada, no sin arrastrar parte de los bacilos que se inyectan por este procedimiento al huésped.

Como es natural, la excitación producida por el hambre obliga a la pulga a buscar con frecuencia el alimentarse; pero a pesar de todos sus esfuerzos no consigue sino lavar la parte anterior del tubo digestivo.

En este estado la pulga resiste un tiempo variable, pero siempre dependiente de la temperatura, y sobre todo del grado higrotimétrico, ya que sin poder injerir líquidos son muy poco resistentes a la sequedad. Durante este período puede propagar la enfermedad, hasta que acaba por morir, o en los esfuerzos desesperados para que la sangre llegue a su estómago puede ocurrir que el bloque de bacilos se rompa y que se forme un canal mdiano o lateral, por el que la sangre pueda penetrar en aquél.

Estas pulgas, que han logrado romper el bloque, son llamadas semibloqueadas, y son las más peligrosas. primero, porque su vida es bastante más prolongada que la de las pulgas enteramente bloqueadas, y segundo, porque en este período, gracias al canal abierto, puede la sangre atravesar el proventrículo; pero éste ha dejado de funcionar como válvula por encontrarse sus espinas completamente aglomeradas en una masa por los bacilos, y la sangre no pasa sino parcialmente al estómago, mientras otra parte muy importante se regurgita.

En 1925, Ricardo Jorge emitió una hipótesis, basada en el fenómeno del bloqueo observado por estos autores, acerca de la estructura del proventrículo en relación con la transmisión de la peste, que fué aceptada por Hirst, según hace constar en el año 1927. Según ella, el hecho de que algunas especies de pulgas no sean infectantes, estribaría en la conformación de su intestino, ya por tener un orificio esofágico demasiado ancho o por su abertura gástrica ensanchada, o bien por los procesos espinosos del proventrículo menos numerosos y menos largos, lo que ocasionaría que la pulga, a pesar de picar y volver a picar sobre un animal infectado, no llegaría a formar el bloque de bacilos o tendría más facilidad para romperle.

Esta hipótesis, que por falta de datos anatómicos suficientes ha sido aceptada como verosímil, se basa en las observaciones clásicas de Bacot y Martin.

Con objeto de buscar una comprobación a esa hipótesis, emprendimos un estudio del proventrículo en las diferentes especies de pulgas de que podíamos disponer. Nuestro intento fué el estudiar de una manera detallada las características anatómicas de dicho órgano, practicando sistemáticamente un gran número de disecciones de pulgas, y además intentar ver las modificaciones que dicho proventrículo sufría por la repleción de sangre, y por una posible infección por bloque. Como no nos era dado observar pulgas espontáneamente infectadas, intentamos hacer una infección experimental con el germen más vecino al bacilo pestoso, o sea con el *B. pseudotuberculosis rodentium*.

Técnica empleada para los ensayos de la infección experimental.

Para hacer las experiencias de infección con el bacilo de la pseudotuberculosis hemos mandado hacer unos frasquitos de cuatro por dos centímetros, cuya boca, muy ancha, va tapada con tela metálica muy fina o con gasa de seda para impedir el paso a su través de los Afanípteros. En cada uno de ellos se coloca un trocito de papel de filtro para permitir a las pulgas reposar sobre él.

En cada uno de estos frasquitos se introduce un lote de pulgas y se los rotula convenientemente, conservándolos en un armario-estufa a temperatura adecuada, que estimamos en 18-20° C, y con humedad suficiente para que puedan vivir el mayor tiempo posible.

Para alimentar a las pulgas se adosan aquéllos a un dispositivo especial, que consiste en un anillo metálico de diámetro ligeramente mayor que los frasquitos, que lleva lateralmente dos asas, en las que se engancha una cinta de goma que abraza la cintura del cobaya, y al propio tiempo sujeta el frasco, Una vez atado el cobaya a una tabla en decúbito supino se le rasura la parte abdominal y se le ajusta la cinta de goma, enganchándola al anillo metálico. En éste se introduce el frasco con la boca hacia abajo para que quede en contacto con la piel del animal, dejándole durante diez a quince minutos de preferencia a oscuras para que piquen. Una vez que han transcurrido, se retira el frasquito, limpiando la boca con un algodón empapado en éter, o si está tapado con tela metálica, flameando ligeramente ésta para desinfectar los excretas que las pulgas expulsan al nutrirse, teniendo cuidado en ambos casos de que los insectos no sufran. Se anota en un cuaderno la fecha en que han realizado la toma de sangre infectante y se traslada el frasco al armario para su conservación, tomando nota de las veces sucesivas que toman alimento.

Con objeto de diagnosticar rápidamente la infección del proventrículo en la pulga, Bichkov y Borzenkov han descrito una técnica, que ha sido la que nosotros hemos seguido en los ensayos de transmisión del bacilo seudopestoso. La técnica en sí es la siguiente:

a) Se mata al insecto con cloroformo.

b) Se le sumerge en alcohol de 90° durante un minuto para desinfectarlo.

c) Se diseca bajo una lupa de diez aumentos, sacando el aparato digestivo por delante, sobre un porta, con una gota de solución salina estéril.

d) Se examina al microscopio con ocular 4 y objetivo 3. Cuando está infectado, el proventrículo presenta un aspecto más oscuro y tiene mayor tamaño que el normal, estando la estructura alveolar de sus paredes aplastada por la presencia de los bacilos.

e) Siembra en masa del proventrículo sobre placa de agar. Por este método se pueden eliminar los ejemplares cuyo proventrículo no presente los caracteres que hemos descrito, y su sencillez permite fácilmente examinar 100 ejemplares diarios, permitiendo asimismo el diagnóstico de una epidemia retrospectiva.

Digamos ya desde un principio que la transmisión experimental de la infección del bacilo seudotuberculoso por medio de las pulgas no nos ha sido posible hasta ahora, y por tanto, nuestros ensayos han sido negativos desde este punto de vista.

El estudio del proventrículo se ha realizado por dos métodos, ya por simple disección y extracción del aparato digestivo de la pulga, ya sea por cortes histológicos coloreados.

Técnica seguida para el estudio de la estructura del proventrículo.

Aunque al principio las pulgas eran estudiadas solamente en fresco, es decir, al poco tiempo de haber matado la pulga, hemos podido comprobar que sirve a nuestros fines cualquier pulga, ya que el revestimiento quitinoso del proventrículo impide que éste sufra durante la putrefacción que necesariamente alcanza a otros tejidos.

Si la pulga está en seco, hay necesariamente que hervirla en potasa al 5 por 100 durante unos minutos, los suficientes para que se ablande, y una vez obtenido el punto necesario, se observa a la lupa binocular el lugar ocupado por el proventrículo, por si éste se hubiera desplazado con la cocción. Una vez que se ha fijado su posición, no hay más que cortar la cubierta quitinosa del exosqueleto, por el segmento anterior y el posterior y oprimir levemente sobre un portaobjetos para que el proventrículo salga al exterior englobado con algunas túnicas externas; pero enseguida se despoja de ellas, y se procede a abrirlo, que es la labor más delicada. Hay que abrirlo longitudinalmente y procurando que el corte sea lo más recto posible, de manera que pueda distinguirse bien las filas de espinas, que son más apretadas y de espinas más cortas y débiles en la parte anterior, mientras que por detrás son más largas y están algo más distanciadas.

Si se ha extraído con cuidado el proventrículo y, sobre todo, si el corte ha estado bien dado, entonces se puede ver con suma facilidad la forma, el grosor y la longitud de las espinas, con lo cual, unido a su separación en el proventrículo, tendremos los datos para comparar los de las distintas especies y poder sacar conclusiones.

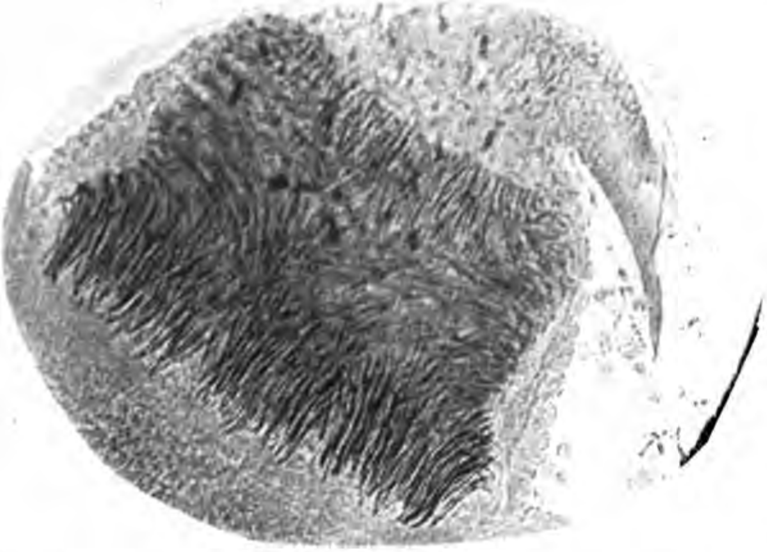
Creemos superior este método al del corte histológico, que también hemos practicado, para observar la preparación en su conjunto y darnos cuenta de la exacta distancia que media entre las espinas, así como de la forma de éstas, aunque el método de los cortes nos ilustra acerca de su posición exacta en el animal en vivo, para lo cual es insubstituíble. Creemos, por tanto, y así lo hemos hecho en nuestro trabajo, que deben usarse ambos procedimientos, que se complementan de manera perfecta para hacer un estudio acabado de la estructura del proventrículo y del recubrimiento interno de espinas.

Estructura del proventrículo.

Visto en corte longitudinal (véase fig. 2, lám. 8) el proventrículo, se advierte que debajo de su superficie externa hay una serie de fibras musculares circulares que sirven para contraer su abertura y, por lo tanto, aproximar las espinas que lleva en su superficie interna, haciendo cerrar el paso

LAMINA 7

1



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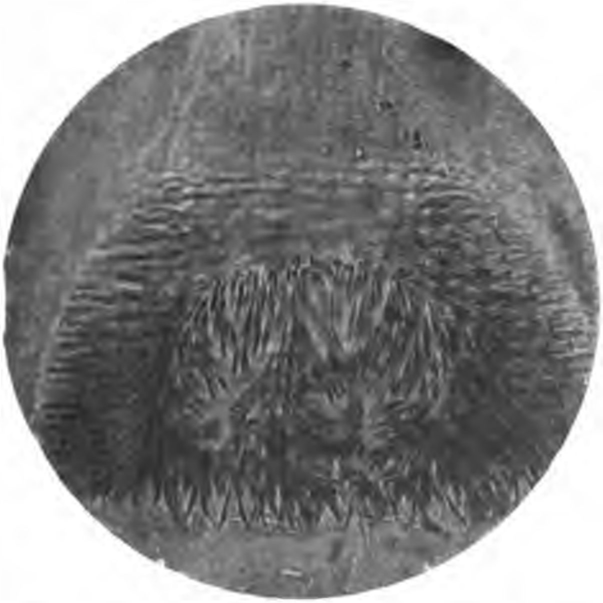
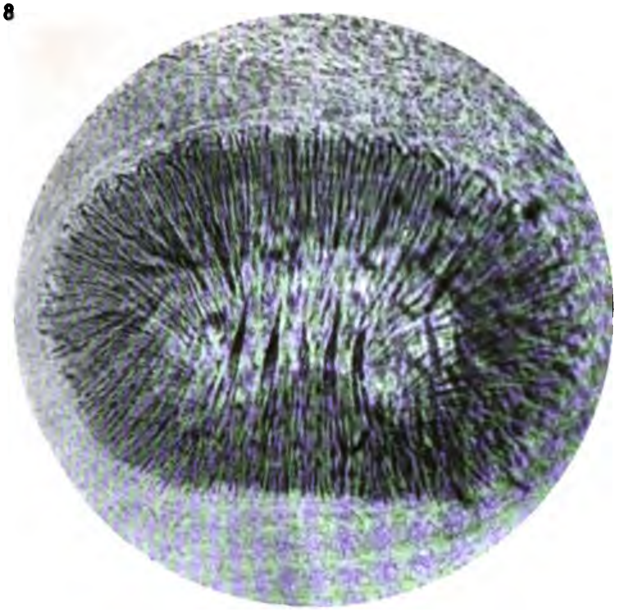


1. Parte posterior del esófago y proventrículo de *X. cheopis*, en el que aparecen las espinas en su posición típica. 2. Aspecto del proventrículo, en el que aparece la estructura aparentemente alveolar a que aluden Patton y Evans.

(Microfotografías *Cartañá*.)

LÁMINA 8



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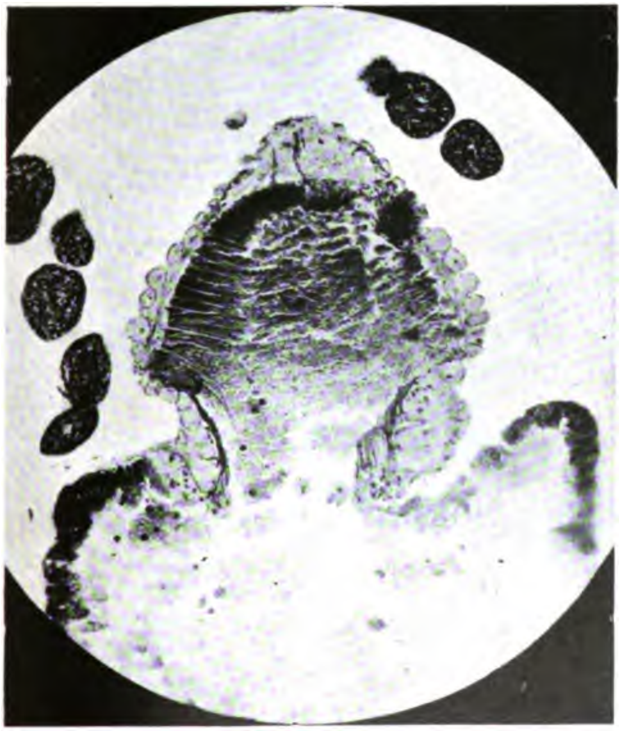

2

1. Proventrículo de *X. cheopis*, visto por la cara anterior, mostrando los procesos espinosos entrecruzados. 2. Corte del proventrículo de la misma especie, en sentido longitudinal, mostrando la capa muscular sobre la membrana quitinosa en la que están insertas las espinas. Se distinguen claramente varias de éstas y la amplia entrada del estómago.

(Microfotografías *Cartañá-Amell.)*

del esófago a dicho órgano para impedir el retroceso de la sangre. Debajo de esa capa muscular hay una membrana quitinosa que está provista de un amplio anillo de espinas quitinosas muy densas. En su borde anterior, las espinas son muy cortas y van precedidas de dos o tres círculos de pequeños abultamientos que quizá representen espinas rudimentarias. Todas las espinas forman círculos, unos a continuación de otros, y están dispuestas con regularidad, inclinadas ligeramente las de cada círculo en relación con las de los siguientes. Todas ellas son curvas, están oblicuas respecto a la superficie del proventrículo y dirigidas hacia atrás.

En cuanto a su forma, hemos de advertir que discrepamos profundamente del criterio de Patton y Evans, cuyos autores afirman que son redondas, aunque parecen hexagonales en su sección, por estar muy cerca unas de otras. En cambio, nosotros, siguiendo la técnica que se ha detallado más arriba y abriendo los proventrículos para examinarlos con más exactitud y sin que la abundancia de procesos espinosos dificulte la visión, hemos podido comprobar que no sólo lo son, sino que su base aparece como una estrella de seis radios, debido a los salientes o quillas que aquéllas presentan. Sólo hay una serie de espinas, precisamente la que ocupa el círculo anterior, que tiene la base en general redondeada.

Hemos de advertir que el número y las dimensiones de las espinas suelen ser diferentes según los ejemplares; en los grandes, con proventrículo grueso, son más numerosas y grandes; en cambio, en los de menor talla son menos numerosas y de algo menor tamaño.

En cuanto al sexo, no hemos podido encontrar diferencias apreciables; si acaso, hemos de indicar que el proventrículo de los machos es ligeramente más pequeño, debido a su menor talla, y lleva por ello menos espinas. A continuación exponemos una ligera descripción de estos órganos en las cinco especies que predominan en las ratas de la población de Barcelona:

X. cheopis.—La sección de las espinas muestra seis salientes, de los que cuatro son más marcados que los otros, dando a la espina una forma parecida a la de una H, de ramas cortas, llevando en la mitad de cada una de éstas una quilla mucho menos marcada. La talla de las espinas es bastante grande; son gruesas, con su extremo romo y algo encorvado. Las espinas son bastante numerosas. En el macho son ligeramente más aserradas que en la hembra, en la cual la denticulación de las quillas es extremadamente delicada.

C. fasciatus.—Tiene la sección de las espinas más regular, con seis quillas bien salientes y casi iguales, no teniendo la forma de H tan marcada como las de la especie anterior. Son de mayor longitud, más finas y más numerosas. Esta especie, como la anterior, tiene los aquillamientos ligerísimamente denticulados, de tal manera que hace falta verlas con inmersión para poder darse cuenta de su existencia. En su extremo son algunas romas, y la mayor parte puntiagudas. En el macho parecen ser más débiles que en la hembra, y algunas entre las puntiagudas tienen un engrosamiento mediano que está separado por una contracción de la parte apical de la espina.

Ct. segnis.—Las espinas de la primera serie son cortas y de base casi circular, apareciendo las de la tercera serie poligonales, pero sin quillas demasiado marcadas; en la cuarta aparecen éstas netamente, en número de seis, asemejándose a las de la *X. cheopis* y teniendo a veces dos quillas muy pequeñas en la rama mediana de la H que modifica su aspecto.

En cuanto a su longitud, son más pequeñas y más gruesas que las de *C. fasciatus,* y unas son agudas, mientras otras tienen el extremo redondeado, y en su mayor parte encorvado.

Ct. felis.—En esta especie las espinas del proventrículo se caracterizan por tener las quillas netamente aserradas, con dentículos que se distinguen muy bien con el objetivo seis. En cuanto a su sección, es bastante parecida a la de la *X. cheopis,* en las espinas de las series centrales, mientras que la primera tiene su base redondeada, y la segunda con un hexágono de vértices un poco afilados.

Ct. canis.—Las espinas son largas, finas y encorvadas en el extremo. Su base es como la de *X. cheopis* pero las dos quillas extremas son más desarrolladas y aparentes. Aunque estas quillas son visiblemente aserradas, no lo son tan fuertemente como en la especie anterior, pero siempre es la denticulación bastante más marcada que en las restantes especies.

¿Puede basarse en la forma y número de las espinas de las diferentes especies algunas conclusiones acerca del por qué se verifique el bloqueo en algunas de ellas de preferencia sobre las otras? A nuestro juicio, ni las diferencias de forma ni de cantidad de espinas observadas en las cinco especies de pulga de la rata nos autoriza claramente a emitir ninguna conclusión.

Por otra parte, parece natural y lógico que en las especies cuyos salientes, dentículos, etc., sean más numerosos proporcionarán a los bacilos más sitios en los que albergarse y en los que poder proliferar. En este caso, habría sin duda que pensar que las espinas aserradas de los *Ctenocephalides* son las más aptas para albergar dichos bacilos, lo cual está en contraposición con el hecho demostrado de que la *X. cheopis* es la que más relación tiene con la propagación de la peste. Sin embargo, acaso sea el corto número en que las especies de aquel género parasitan a las ratas lo que impide que lleguen a tener importancia desde el punto de vista de la epidemiología de esa enfermedad, a menos de admitir la explicación que nosotros damos a este problema más adelante.

En cuanto al *C. fasciatus,* sus espinas, con las quillas menos marcadas que en *X. cheopis,* parece que pudieran indicar una menor facilidad para la propagación de los bacilos, lo que pudiera estar contrarrestado por su número marcadamente mayor.

Hasta aquí nuestras observaciones y las deducciones de este estudio, que acogimos con todo cariño, siguiendo la idea trazada por el Dr. Ricardo Jorge al exponer su teoría. Como última conclusión, creemos que no bastan la disposición ni la forma de las cerdas del proventrículo para explicar por sí solas el distinto poder infectante que tienen las especies españolas que habitan sobre la rata, aunque sí contribuirían a apoyar quizá otros hechos, como son el poder bactericida de los jugos intestinales de estas pulgas, que indudablemente han de variar en las distintas especies, ya que el poder fagocitario de la sangre ingerida habría de ser forzadamente la misma en las diversas especies a igualdad de las otras condiciones, como temperatura, humedad, animal de que procede, etc.

Posible influencia, en el desembarazo del proventrículo infectado, de la alimentación alternativa de una pulga sobre varios animales, cuando uno de ellos es inmune a la peste.

Acabamos de indicar que en nuestro estudio acerca de la anatomía y de la estructura del proventrículo no hemos encontrado una base cierta para explicar el papel más o menos importante que juegan las distintas especies de pulgas en la propagación de la epidemia pestosa. Ello nos ha sugerido que quizá, además de la acción bactericida más o menos intensa de los jugos digestivos de la pulga, tenga importancia el huésped habitual de ésta, para determinar hasta qué punto sea peligrosa la especie.

En efecto, es sabido que la sangre ingerida por la pulga tiene por sí misma un poder fagocitario acentuado, que hace que muchos bacilos pestosos queden destruídos por completo. Esta acción fagocitaria de la sangre varía mucho según las distintas especies de animales, y aun en algunas probablemente la inmunidad que presentan con respecto a la peste debe estar regulada por esta acción destructiva y fagocitaria de su sangre.

Si una especie de pulga, cuyo huésped habitual sea refractario y cuya sangre tenga un gran poder fagocitario se nutre sobre un animal infectado, por ejemplo, una rata pestosa, es indudable que si después cambia de huésped, volviendo al primero, la sangre nuevamente ingerida ha de actuar sobre los bacilos que se almacenaban en el interior de su tubo digestivo y ha de destruir gran parte de ellos o quizá todos.

A esta acción coadyuva el que los cambios de huésped se traduzcan en digestiones más largas y difíciles, como han indicado numerosos autores.

Ciñéndonos a un ejemplo concreto, el *Ctenocephalides,* cuyas especies son habituales en el perro, a pesar de tener su proventrículo bien poblado de espinas y de que las quillas de éstas son marcadamente más aserradas que en los otros géneros, condición, a nuestro juicio, favorable para la pululación de los bacilos, puede, después de infectarse sobre una rata pestosa, limpiarse de bacilos al volverse a nutrir sobre su huésped predilecto, por la acción fagocitaria de la sangre del perro, que, como es sabido, es resistente a la infección pestosa.

Así, este afaníptero, a pesar de su estructura proventricular favorable para el desarrollo de los bacilos, puede tener una importancia mucho menor que otras especies con peores condiciones para éstos.

A esta hipótesis, que damos como probable, aun cuando no hayamos podido comprobarla, conduce la necesidad de buscar una nueva orientación que nos explique por qué especies de pulgas que tienen una estructura orgánica parecida y que pueden infectarse con igual facilidad en condiciones experimentales, juegan un papel muy diverso en la epidemiología de la peste.

CAPITULO VI

INFECCION PESTOSA DE LAS RATAS

Describiremos someramente esta infección, dividiendo el capítulo en los siguientes puntos:

A) Infección natural.

- **I.—Peste aguda.**
- **II.—Peste crónica.**
- **III.—Sin lesiones viscerales.**

B) Infección experimental.

- **I.—Vía subcutánea.**
- **II.—Vía intraperitoneal.**
- **III.—Vía cutánea.**
- **IV.—Vía digestiva.**
- **V.—Vía mucosa, etc.**

C) Diagnóstico.

A) INFECCIÓN NATURAL.—I. Peste aguda

Signos macroscópicos "post-mortem".

En la infección natural se dan los siguientes signos:

a) Bubón cervical (75 por 100 de las ratas pestosas).

b) Apariencia granulosa del hígado, debida a la presencia de pequeñas áreas de necrosis esparcidas sobre superficie. (Este signo fué observado en 55 por 100 de las ratas en Bombay por la Comisión inglesa.)

c) Congestión subcutánea general.

d) Hemorragias, ya en el tejido subcutáneo, ya en los órganos.

e) Exudado pleural claro abundante.

f) Bazo consistente (gránulos en 4, 5 por 100 de las ratas).

g) Pulmones gránulos necróticos en 2, 3 por 100.

h) Peste neumónica primaria en 0,6 por 100 de las ratas.

En 36 ratas *(E. Norvegicus)*, infectadas experimentalmente por la Comisión inglesa de la peste en la India ,los signos más constantes observados fueron:

a) Congestión subcutánea.

b) Aspecto granuloso del hígado.

c) Líquido pleural claro.

Los tres signos fueron presentados por la mayor parte de las ratas, y ninguna dejó de presentar ***uno*** o más de ellos.

Incluso en estado de putrefacción avanzada, pueden conservar estos tres signos:

Bubón primario.
Exudado pleural.
Aspecto granular del hígado.

II. Lesiones de peste crónica

Las principales lesiones son:

1.ª Bubón.
2.ª Zonas necróticas en bazo.
3.ª Abscesos crónicos en bazo o hígado.
4.ª Periesplenitis.
5.ª Cicatrices en el bazo.
6.ª Bazo dicotomizado.
7.ª Adherencias del bazo con tejidos vecinos.

La Comisión inglesa prefiere substituir el término de "Peste crónica" por el de "resolvilng" o "resolved" (peste en vías de resolución o regresión), que implica la idea de un proceso con tendencia a la curación, llegando a la esterilización y al tejido cicatrizado.

La mayor parte de las ratas con estas lesiones se encuentran ***durante o inmediatamente después*** de una epizootia de peste. Estos hechos han podido ser reproducidos experimentalmente.

III. Peste en ratas sin lesiones viscerales

En este caso no presentan ningún signo macroscópico, y la infección se descubre únicamente por inoculación al cobaya. Como se desprende, esta forma de infección pasa desapercibida cuando solamente se estudian aquellas ratas que ofrecen macroscópicamente a la autopsia alguna sospecha de peste, siendo necesario para darse cuenta de ellas la inoculación sistemática a cobayas con vísceras de todas las ratas que se autopsian; esto no puede hacerse en todos los servicios, si bien puede disminuirse el gasto, el trabajo y el local para animales inoculados que esto implicaría, inoculando un cobaya por cada lote de 10 ratas por eje. Las formas septicémicas con pocos o sin signos macroscópicos son más bien propias del comienzo de la epizootia en localidad virgen. Las formas crónicas o "resolving" se encuentran, por el contrario, al final de una epizootia.

B) INFECCION EXPERIMENTAL

I. *Vía subcutánea.*—Como en el cobaya, la inoculación de una pequeñísima cantidad (aguja cargada de virus) produce la muerte en un período de dos a cinco días, presentando edema rosado en el sitio de inoculación; ganglios hipertrofiados y congestionados, con zona hemorrágica alrededor; órganos abdominales congestionados, bazo *voluminoso* con erupción de pequeños tubérculos miliares; en pleura y peritoneo existe un poco de serosidad conteniendo el bacilo; las suprarrenales pueden estar congestionadas; *frotis* de vísceras con gran número de bacilos, pero menos abundantes en la sangre. Si la infección dura, por ejemplo, ocho días, se observa a veces un cordón linfático desde el punto de inoculación, que va a terminar al ganglio inguinal correspondiente; lesión local caseificada fuertemente adherida a la pared abdominal; aspecto de lesión tuberculosa con pus caseoso; gran edema gelatinoso; adherencias de los intestinos a la lesión local; congestión hemorrágica del tejido celular subcutáneo; grandes lesiones viscerales; *hígado* con grandes lesiones tuberculosas; adherencias de la pared abdominal con estómago, etc.; bazo hipertrofiado, sembrado de tubérculos; algún ganglio mesentérico. En el *tórax*, los pulmones sembrados de grandes tubérculos, que ocupan toda la víscera, de tamaño de media lenteja. Estos tubérculos tienen una zona hemorrágica difusa alrededor; líquido pleural claro, no muy viscoso.

II. *Vía intraperitoneal.*—Muerte en veinticuatro a cuarenta y ocho horas; puede presentar pequeño ganglio de la zona correspondiente, si dura cuarenta y ocho horas; congestión hemorrágica alrededor del sitio de inoculación. Abierto el peritoneo, se observa gran congestión de los intestinos y gran cantidad de exudado peritoneal viscoso, hilante y riquísimo en gérmenes que ofrecen su aspecto típico de coloración bipolar, cantidad de gérmenes como si fuera un cultivo puro. El líquido tiene una *viscosidad tal,* que al practicar el aislamiento del germen sobre placas casi siempre el cultivo crece en la primera mitad de la placa correspondiente al sitio donde se han dejado la semilla, y raramente ya pasa cultivo a la segunda, y mucho menos a la tercera placa, a pesar de haber repartido bien dicha semilla con la varilla de vidrio.

III. *Vía digestiva.*—Es también posible. La Comisión inglesa de la peste no la considera muy frecuente, si bien la observó y consignó experimentalmente. La Comisión alemana ("Rapport", 1889) describe que la obtuvo sin dificultad alimentando a ratas en cuyo alimento se había puesto una gota de cultivo virulento, o con cadáveres de ratas pestosas. Producían la muerte uniformemente en dos a tres días, y describen tres tipos de lesiones *post-mortem:*

a) Más comúnmente hipertrofia y congestión de ganglios submaxilares y suprahioideos, con el cuadro general septicémico.

b) Con menos frecuencia, infección primaria del estómago e intestino, numerosas hemorragias puntiformes alrededor del píloro, hinchazón e infiltración hemorrágica de los folículos linfáticos y ganglios mesentéricos, los cuales estaban bastante hipertrofiados y contenían bastantes bacilos.

c) Bastante frecuentemente, neumonía por aspiración, mostrando los pulmones focos inflamatorios de tamaño variable, conteniendo numerosos bacilos, hígado congestionado, bazo hipertrofiado.

La Comisión inglesa establecía dos principales diferencias *entre la infección natural* en las ratas y la *infección por vía gástrica:*

a) En la infección natural, el bubón está en el cuello.

En la infección por vía gástrica, bubón mesentérico.

b) En la infección natural, no se presentan cambios patológicos ni en el estómago ni en el intestino.

En la infección por vía digestiva, estas lesiones son ostensibles (hemorragias en las paredes del estómago, congestión de las intestinos y, sobre todo, hipertrofia de las placas de Peyer).

IV. *Vía mucosa.*—Muy sensible. La inoculación en la mucosa nasal y conjuntival mata en tres o cuatro días, con lesiones parecidas a las encontradas en la infección por vía oral.

V. *Vía cutánea.*—Aparece una reacción inflamatoria alrededor; marcada por un ligero enrojecimiento y formación de pústulas umbilicadas (con bacilos pestosos abundantes). Después de pocos días se hipertrofian los ganglios linfáticos, y la muerte ocurre a los cuatro o cinco días. Los signos post-mortem son parecidos a los de la inoculación subcutánea.

Según la Comisión inglesa, este procedimiento falla en el 2 por 100 de los casos con material reciente, y en el 10 por 100 de los casos con ratas en putrefacción.

Diferencias entre las lesiones producidas por la infección natural en la rata y la infección experimental en el cobaya.

COBAYA	RATA
Raras hemorragias subcutáneas.	Frecuentes hemorragias subcutáneas.
Edema general subcutáneo característico.	Raro.
Hemorragias subserosas intestinales comúnmente.	Raras.
Organo importante para el diagnóstico: *bazo.*	Organo importante para el diagnóstico: *hígado.*

Posible y variable inmunidad de las ratas a la peste.

Los estudios hechos en este sentido, y que citamos al hablar de los factores que explican la desaparición espontánea de una epizootia han demostrado que las ratas pueden adquirir cierta inmunidad contra el *b. pestis* y que pueden transmitirla a las nuevas generaciones.

C) DIAGNÓSTICO DE LA PESTE EN LAS RATAS

En vida.—La salida de las madrigueras en pleno día, la marcha ebria y el dejarse capturar fácilmente constituyen los principales signos.

Signos "post-mortem".—Los anteriormente descritos. Ellos dan el diagnóstico de sospechosa. Confirman luego el *diagnóstico definitivo* las pruebas de laboratorio: frotis, cultivos, inoculaciones, reacción de Ascoli.

Causas de error:

a) FORMAS INVOLUTIVAS.—En los frotis no siempre se observa la forma típica de los bacilos, pues las lesiones crónicas contienen muchas veces formas atípicas (f. de involución).

b) GÉRMENES CON COLORACIÓN BIPOLAR.—Se encuentran también muy frecuentemente en estos frotis gérmenes gramnegativos con coloración bipolar, muy parecidos al bacilo pestoso; son gérmenes de la putrefacción intestinal, que pueden inducir fácilmente a error si no se hacen también las pruebas subsiguientes de cultivo e inoculación. Sabido es también que el colibacilo invade *post-mortem* las vísceras, presentando también la forma de cocobacilo gramnegativo con coloración bipolar. A este efecto, nosotros hemos podido comprobar, haciendo una serie de autopsias y siembras escalonadas de ratas que habían sido muertas al mismo tiempo con el CNH, que al cabo de una hora de la muerte se encuentra ya el colibacilo en las vísceras de las ratas. Es posible que la asociación de los gérmenes de la putrefacción disminuya la virulencia del bacilo pestoso.

La presencia de estos gérmenes (el bacilo *proteus* sobre todo) entorpece el aislamiento por cultivo. Para obviarlo puede hacerse el aislamiento sobre medio de Endo o agar cristal violeta al 1/70.000.

El aislamiento en cultivo es directamente proporcional a la reciente muerte del animal examinado. Interviene también principalmente la temperatura ambiente; así, por ejemplo, ha podido ser aislado el bacilo después de noventa y tres días a 5°, pero solamente después de cuatro días a 32°.

La inoculación al cobaya.—Con vísceras de una rata muerta no debe hacerse nunca la inoculación por vía intraperitoneal, por las mismas razones dichas. Se hará por vía hipodérmica, y si está en putrefacción, por vía cutánea.

Reacción de Ascoli (precipitinas).—Si el animal a estudiar está ya en plena putrefacción, puede practicarse la reacción de Ascoli.

La técnica aconsejable es la siguiente:

1. Triturar vísceras en putrefacción.
2. Añadir de cinco a 10 partes de solución salina.
3. Hervir varios minutos.
4. Filtrar repetidas veces (en frío) hasta obtener líquido claro.
5. Añadir este filtrado gota a gota a un tubo que contiene suero antipestoso (el líquido que se añade deberá resbalar por las paredes del tubo y así los dos líquidos, suero y filtrado, se superponen). La reacción positiva da un anillo de precipitado.

Deben añadirse cuatro tubos de control:

1. Suero anti + solución salina = Negativa.
2. Suero anti + extracto de cultivo de bacilo pestoso = Positiva.
3. Suero normal + extracto de órgano sospechoso = Negativa.
4. Extracto de órgano sospechoso solamente = Negativa.

Lesiones viscerales de las ratas parecidas a la peste

Tularemia.
Tripanosomiasis.
Hepaticola Hepática.
Quistes hepáticos producidos por la "Tænia crassicolis".
Seudotuberculosis.

Tularemia.

Enfermedad infecciosa de conejos y ardillas, frecuente en Tulare (California). Parecida a la peste.

En el animal.—Septicemia aguda mortal asociada con hipertrofia de los ganglios y del bazo, con presencia de nódulos blanquecinos necróticos en bazo e hígado. En el bazo son muy abundantes, pareciéndose a la tuberculosis generalizada.

Germen gramnegativo, inmóvil, cocobacilo muy pequeño, de 0.7 a 0,8 micras. *No cultiva en agar común.* En el hombre produce una enfermedad parecida a la peste, si bien más larga y letalidad menor.

DIAGNÓSTICO.—Cultivos, aglutinación, inoculación al cobaya.

Tripanosomiasis.

El *Tripanosoma levisi* puede dar en las ratas lesiones parecidas a la peste; el diagnóstico se establecerá fácilmente por el hallazgo del tripanosoma *en la sangre* (gota gruesa, sangre del corazón).

Hepaticola hepática.

Es un gusano nematode de la familia de los trichuridos, cuya hembra pone los huevos en el hígado de las ratas y les produce unas lesiones de tipo cirrótico en el hígado, que pueden inducir a error. El diagnóstico con la peste lo estableceràn:

Frotis negativos, cultivos negativos, inoculaciones negativas, cortes histológicos demostrando la presencia en número considerable de los huevos del parásito.

Después de haberlas visto unas cuantas veces, estas lesiones se diagnostican perfectamente a simple vista.

Quiste hepático producido por la "Tænia crassicolis".

La larva de este parásito empieza a desarrollarse en el hígado de la rata y da lugar a un *quiste blanquecino* conteniendo el parásito y que sobresale de la convexidad del hígado de la rata.

Seudotuberculosis de los roedores.

Es esta enfermedad la que sin duda alguna se parece más a la peste, tanto por sus lesiones como por las características de su germen causal, "Bacilo pseudotuberculosis rodentium", llamado también *Bacilus pseudopestis* por su semejanza con el pestoso.

Es frecuente la infección natural de los roedores por este germen, y en las crías de cobayas se observan a menudo epizootias graves, que en alguna ocasión han podido hacer nacer la sospecha de la infección pestosa.

En los exámenes sistemáticos de las ratas con vista a despistar una posible epizootia pestosa debe, pues, tenerse en cuenta siempre aquella infección. Es de importancia además el hecho de que el cobaya que proceda de un foco infectado de seudotuberculosis puede morir por esta infección después de ser inoculado con otro material patógeno y falsear así la interpretación de momento.

Las lesiones producidas por el *Pseudotuberculosis rodentium* en el cavia y en las ratas son nódulos o tubérculos redondeados blancogrisáceos, de 2 a 3 mm. de diámetro, de un contenido caseoso o cremoso, y principalmente esparcidos por el hígado, bazo y pulmones. Dichos tubérculos son bien definidos, o sea que el tránsito entre el nódulo y el tejido sano es franco; además, generalmente sobresalen de la superficie; en la peste, en cambio, el tubérculo tiene una zona inflamatoria congestiva alrededor (característica de gran valor diferencial), y además suele ser más pequeño y no tan sobresaliente.

En el contenido caseoso de los nódulos seudotuberculosos se encuentran un gran número de cocobacilos gramnegativos con coloración bipolar.

La infección experimental en el conejo, cobaya o ratón produce la muerte en un período de *una* a *tres semanas,* con formación caseosa en el punto de la inoculación subcutánea y adenitis de los ganglios regionales, además de las lesiones dichas en hígado, bazo y pulmones.

Los bacilos se encuentran en cantidad tanto en la lesión local como en los ganglios.

La infección natural se produce por vía digestiva; de aquí que se observen también a la autopsia adenitis en los ganglios mesentéricos.

El bacilo *Pseudotuberculosis rodentium* pertenece al grupo de las pasteurellas, como el bacilo pestoso, y entre los dos el parentesco y la semblanza son tan grandes, que, como dice Topley, la identificación para una raza dada no es siempre cosa fácil. No describiremos detalladamente las características de cada uno de estos gérmenes, pero sí haremos resaltar aquellas que mejor pueden servir para su distinción. Estas pueden resumirse en:

a) *Diferencias en la movilidad.*—El *Pseudopestis* es *móvil* en los cultivos en caldo crecidos a 20°-22° durante dieciocho horas, mientras que el bacilo pestoso es absolutamente inmóvil (carácter el más útil para la distinción, según Topley).

b) *Diferencias culturales.*—Relativas, sobre todo, al crecimiento en la patata (el pestoso no crece, y el *Pseudopestis* generalmente sí) y al comportamiento con el medio de Petruski, en el cual el *B. pestis* vira al rojo, mientras el *Pseudopestis,* por el contrario, alcaliniza, dándole, por lo tanto, coloración azul.

c) *Poder patógeno para los animales de laboratorio, y en especial para la rata blanca,* a la que el bacilo *Pestis* produce la infección pestosa típica.

y, en cambio, el *Pseudopestis* no, siendo además la rata blanca relativamente resistente a la seudotuberculosis.

d) Pruebas de aglutinación y de precipitación con los sueros respectivos.

Existen además otras seudotuberculosis que no presentan ya, ni por el tipo de la lesión ni por los caracteres del agente etiológico, un parecido tan grande con las lesiones pestosas, tales, por ejemplo, como la seudotuberculosis del ratón, producida por un germen difteroide grampositivo, que lleva el nombre de *Corynebacterium Kutscheri,* y que nosotros hemos tenido ocasión de aislar dos veces en pequeños nodulitos pulmonares de dos ratas pertenecientes a *Epimys Norvegicus.*

Estudio sobre los antígenos del bacilo pestoso y relaciones antigénicas con el B. pseudotuberculosis rodentium.

Deberemos resumir aquí, por considerarlos de alta importancia, los trabajos del profesor Schütze, del Lister Institut de Londres, que hemos tenido ocasión de observar a nuestro paso por aquel centro en mayo de 1933, expresando desde aquí nuestro agradecimiento a dicho profesor y a la Dirección de aquel Instituto.

Las experiencias de Schütze se basan en el hecho observado por Rowland en el año 1914 de que el bacilo pestoso, creciendo a 37°, desarrolla una envoltura gelatinosa demostrable fácilmente por medio de preparaciones con tinta china. Mezclando una pequeña asa de cultivo en agar o del centrifugado de un cultivo en caldo, con igual cantidad de tinta china y haciendo una fina extensión sobre porta, se observa que el germen pestoso crecido a 37° está rodeado de una zona clara de considerable extensión (envoltura gelatinosa), pero no así el germen crecido a 20°, que se muestra desprovisto de ella.

En las razas virulentas de pase se puede observar un débil desarrollo de esta envoltura ya a los 26°, cosa que no sucede con las razas viejas de laboratorio no virulentas, que necesitan la temperatura alta de 37°. De todos modos, a 37° el hecho se observa en todas las razas, sea cual fuere su grado de virulencia.

Un hecho interesante es el de que la formación de esta envoltura gelatinosa *no se observa en el bacilo seudotuberculoso Rodentium,* de tal manera, que, según Rowland, esta característica serviría para la diferenciación de estos dos gérmenes.

Rowland no pudo colorear dicha envoltura, y dice que no puede asimilarse a una cápsula porque no se observa una limitación definida. Posteriormente, los estudios de Schütze, especialmente, han demostrado que dicha envoltura gelatinosa contiene un antígeno completamente definido y diferente del antígeno contenido en el cuerpo del bacilo.

Calentando a 60° una suspensión en solución salina de un cultivo crecido a 37° de *B. pestis* se obtiene una completa disolución de dicha envoltura, y la parte que sobrenada de esta suspensión centrifugada contiene una gran porción de la substancia de esta envoltura y una pequeña cantidad de la substancia somática.

La misma operación (suspensión en ClNa, calentamiento a 60° y centrifugado) practicada con un cultivo crecido a 26° da por resultado que la parte superior contiene una gran proporción de substancia somática y muy pequeña de la substancia envolvente.

Calentando durante un cuarto de hora a temperatura de ebullición el antígeno de la envoltura, se *hapteniza,* o sea que es aún capaz de dar la reacción de precipitación específica con su suero correspondiente, pero es ya completamente inerte como inmunógeno en el cuerpo del animal, o sea que no puede ya producir anticuerpos.

Calentando durante una hora se destruye también su poder de reaccionar *in vitro* específicamente, mientras que el antígeno somático resiste este calentamiento, siendo aún capaz de reaccionar con su correspondiente antisuero.

Así, pues, el antígeno somático es *termoestable,* y el antígeno de la envoltura es *termolábil.*

En otras palabras, este antígeno de la envoltura, calentado a 100°, es rápidamente convertido en un hapteno; pero si el calentamiento a 100° persiste durante una hora, destruye también la propiedad de reacción in vitro.

Con la ayuda de este conocimiento fué posible preparar sueros conteniendo, por una parte, principalmente anticuerpos de envoltura y, por otra parte, sueros con anticuerpos somáticos únicamente.

Así, pues, de este trabajo se desprende que:

1.° El *B. pestis* posee dos antígenos, uno contenido en la envoltura gelatinosa y el otro en la porción somática de la bacteria.

2.° El desarrollo del antígeno de la envoltura es favorecido por el crecimiento a temperatura de 37°.

3.° El antígeno de envoltura es termolábil, siendo haptenizado por calentamiento a 100° durante un cuarto de hora y destruído después de una hora a esta temperatura.

4.° El antígeno somático es más termoestable, siendo capaz de provocar aún anticuerpos después de un calentamiento de una hora a 100° y pudiendo actuar *in vitro* aún después de tres horas a 100°.

Antígenos del Bacilo seudotuberculosis Rodentium y parentesco antigénico con el bacilo pestoso.

a) El bacilo seudotuberculoso Rodentium está dotado de movilidad (propiedad demostrada de nuevo por Arkwright en 1927) y posee dos antígenos: flagelar y somático.

Schütze ha estudiado 18 razas aisladas de aves y de mamíferos y una del hombre, demostrando en ellas tres grupos diferenciables por su antígeno somático, mientras que todos poseen idéntico antígeno flagelar. Las aisladas de aves y mamíferos constituían los grupos I y II, y la raza aislada del hombre difería de las anteriores, constituyendo el grupo III junto con otra raza inmóvil aislada por Krishnamurti en India como el agente causal de una linfangitis bovina, y estudiada por Schütze. Posteriormente ha sido aislado de un "titi" por Miss Rhodes en el Lister Institut otro germen, que ha constituído el grupo IV, puesto que difería de los otros tres grupos del mismo modo que éstos entre sí, o sea por su antígeno somático, siendo idéntico a los demás grupos su antígeno flagelar.

b) Las pruebas de aglutinación y de absorción establecen la identidad del antígeno flagelar en todas las especies y la diversidad del "smooth", antígeno somático en cada tipo serológico. Siendo estos dos antígenos los únicos puestos en evidencia en una prueba de aglutinación cuando los cultivos empleados pertenecen a la variedad lisa.

Ahora bien, en las pruebas de precipitinas, tres antígenos entran en juego de una manera evidente. Este tercer antígeno representa indudablemente el armazón o esqueleto fundamental o básico, "rugoso", del bacilo, el cual, según la teoría de Bruce White, entra en acción en una prueba de aglutinación solamente cuando el germen pierde su parte somática lisa ("smooth"), transformándose en rugoso ("rough"); pero cuando el germen está en el estado "smooth" (liso), está enteramente cubierto por el antígeno específico y excluído así de la participación en la aglutinación el antígeno "rough" contenido en el armazón o parte más central del germen.

En la prueba de precipitación, las condiciones son diferentes, y los tres antígenos están en solución prestos a reaccionar con sus respectivos anticuerpos.

Las experiencias de Schütze sobre este particular (cuyos detalles y técnicas omitimos) le conducen a las conclusiones siguientes:

1. *B. pestis* posee dos antígenos: uno que corresponde a la substancia envolvente y otro a la substancia somática.
2. *B. pseusotuberculosis rodentium* posee tres antígenos: *a*) antígeno flagelar, que es común a los cuatro tipos de las especies; *b*) un antígeno somático, que es específico y puede ser considerado como el "smooth" antígeno somático, y *c*) un antígeno somático que, como el flagelar, es co-

mún a todos los tipos de *B. pseudotuberculosis rodentium* y que es considerado como el "rough" antígeno somático (fig. 5).

El antígeno común al *B. pestis* y al *B. pseudotuberculosis rodentium* es este "rough" antígeno somático, y es en virtud de esto por lo que existe un parentesco serológico entre los dos gérmenes.

4. El *B. pestis* debe ser considerado como existiendo normalmente sólo en el estado rugoso, no existiendo antígeno "smooth".

Valor profiláctico del antígeno somático y del antígeno de la envoltura del bacilo pestoso.

Schütze llevó a cabo este estudio preparando vacunas incubadas unas a 26° y otras a 37°. Diversos lotes de ratas fueron inmunizadas con estas vacunas, y luego sometidas a inoculaciones de cultivos virulentos o emulsiones de bazo pestoso, en cantidad determinada. Los resultados demostraron que el crecimiento a 37° produce una vacuna de un mayor poder profiláctico que el crecimiento a 26°. Así, por ejemplo, en una de las experiencias, de 192 ratas inmunizadas con la vacuna crecida a 37° sobrevivieron a la inoculación de prueba el 72 por 100, mientras que de 143 ratas inmunizadas con la vacuna crecida a 26° sobrevivieron el 32 por 100, y 65 ratas control murieron todas a los cuatro días de dicha inoculación de prueba.

Otra serie de experiencias, en las que fueron empleados diversos lotes de 21 ratas cada uno, inmunizadas con los dos tipos de vacuna obtenida en diferentes medios de líquido, dieron también los resultados siguientes:

De 21 ratas inmunizadas con una vacuna obtenida por un cultivo de 37° y sometidas luego a la inoculación de prueba sobrevivieron el 72 por 100.

Un número igual de ratas inmunizadas con la misma vacuna, pero sometida durante una hora a la temperatura de 100°, murieron todas a los cinco días de la inoculación de prueba.

Otro lote de 21 ratas sometidas directamente a la inoculación de prueba murieron todas a los cuatro días. Así, pues, la vacuna es inutilizada como tal cuando está sometida a este calentamiento, puesto que las ratas inoculadas con ella no ofrecen más resistencia que las ratas control.

Como que, según hemos visto, esta temperatura de 100° hapteniza el antígeno de envoltura, pero no el somático, debe, pues, sacarse la conclusión de que esta substancia envolvente precisamente es la parte constituyente de mayor importancia de la vacuna.

Las conclusiones de estos trabajos del profesor Schütze sobre el poder profiláctico de los antígenos del bacilo *pestis* pueden resumirse en los siguientes:

1.° Tanto las vacunas obtenidas con cultivos en agar como las obtenidas en caldo, aumentan su poder profiláctico en un 50 por 100 por el hecho del crecimiento a 37° en lugar de a 26°. La naturaleza del caldo empleado constituyendo variaciones en la composición del medio de cultivo no afecta para nada el resultado.

2.° Como es precisamente este crecimiento a 37° el que favorece el desarrollo de la substancia envolvente, se deduce que es el antígeno de la envoltura el que es prácticamente importante.

3.° Calentando el cultivo vacuna a 100° durante quince minutos, la vacuna pierde todo su valor. Como que esta temperatura hapteniza la substancia envolvente, mientras que no altera la substancia somática, que se comporta como antígeno capaz de formar anticuerpo después de un calentamiento a 100° durante una hora, se deduce nuevamente que es el antígeno de la envoltura el prácticamente importante.

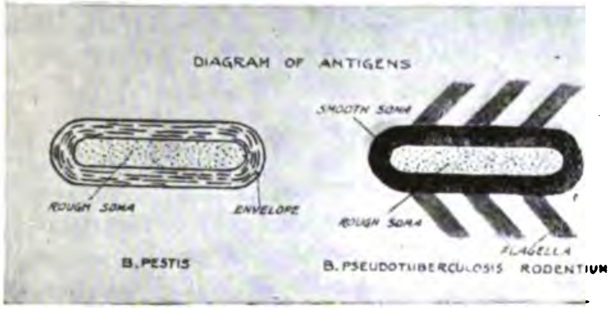

Fig. 5.—Diagrama de antígenos, según Schütze.

4.° Calentada a 60° durante una hora en presencia de 0,5 por 100 de fenol, la vacuna disminuye su poder profiláctico; pero calentando únicamente a 56° y durante media hora, antes de añadir el fenol, no disminuye el poder de la vacuna.

5.° Las vacunas de caldo incubadas a 37°, aunque más potentes, son menos tóxicas que las crecidas a 26°.

Después de resumir estos trabajos de Schütze, creemos pertinente hacer la siguiente observación.

Si el antígeno de valor profiláctico en el *B. pestis* es el antígeno de la envoltura, y, por otra parte, el antígeno común con el *B. pseudotuberculosis rodentium* no es precisamente éste, sino que es el "rough" antígeno, no creemos que tengan demasiado fundamento los ensayos hechos por ciertos autores sobre la vacunación contra la peste por medio del *pseudotuberculosis rodentium.*

CAPITULO VII

RESULTADOS DE AUTOPSIA EN LAS RATAS DE BARCELONA

Conjuntamente con el estudio sobre la entomología de la peste bajo sus dos aspectos de estudio sistemático y experimental, que llevamos descrito, era necesario, como complemento del estudio epidemiológico del brote de peste de 1931 que describiremos luego, hacer una investigación relativa a la epizootia en las ratas. Para esto hemos practicado la autopsia de un número de 4.268 ratas capturadas en las condiciones que describimos en otro lugar; cada ratera lleva una nota con las señas del sitio de captura, de manera tal, que cada rata examinada queda anotada en nuestra hoja de trabajo con el sitio exacto de su procedencia.

Las observaciones que vamos a resumir, relativas a esta parte de nuestro trabajo, se referirán a:

a) Peste.
b) Lesiones de *Hepaticola hepática*.
c) Quiste hepático de la *Taenia crassicolis*.
d) Lesiones producidas por el *Bacilo pseudotuberculosis Kutscheri*.
e) Espiroquetosis icterohemorrágica.

a) Infección pestosa.—Los resultados han sido negativos en cuanto a peste; únicamente en el comienzo de este estudio, y durante la epidemia de 1931, tuvimos ocasión de observar una rata (procedente del Torrent de la Guineu, donde se habían dado cuatro casos de peste humana), que presentaba lesiones macroscópicas de peste en sus vísceras, y cuyos frotis demostraron la presencia de numerosos bacilos gramnegativos con coloración bipolar, de morfología idéntica a la del *B. pestis;* sin embargo, como la inoculación del bazo al cobaya dió resultado negativo, no podemos afirmar que se tratara de peste, pero tampoco podemos deducir la conclusión negativa, tratándose de una rata muerta, y por las razones que indicaremos luego, relativas a la escasa vitalidad del bacilo pestoso en las ratas en putrefacción cuando la temperatura ambiente es alta, como lo era (agosto de 1931).

En las demás ratas observadas no hemos encontrado bacilo pestoso. Debemos indicar, sin embargo, después de lo dicho referente a las causas de error y la presencia de infecciones ***sin lesiones macroscópicas,*** que el procedimiento que nosotros hemos seguido, al igual que el que se sigue en todas las encuestas de este orden, incluso en aquellas que, por sus estudios y descubrimientos nos dieron casi todos los conocimientos que actualmente poseemos sobre esta infección, adolece de un defecto capital, y es el que, como solamente se practican siembras e inoculaciones de aquellas ratas que ya son sospechosas por sus caracteres macroscópicos, pueden pasar por alto los casos que ofrezcan estas lesiones discretas o que no las presenten en absoluto, no obstante ser pestosas.

Y tanto es así, que nuestro primer intento al presentar el programa de nuestra encuesta a la Comisión Permanente de Investigaciones Sanitarias era el de practicar esta investigación sin frotis ni siembras, sino por inoculación directa y sistemática al cobaya del producto del triturado del hígado o bazo de lotes de cinco o de diez ratas. Pero al intentar ponerlo en práctica y ver que la autopsia de 25 ó 30 ratas diarias implicaba el inocular tres o seis cobayas cada día y tener al cabo de pocos días 50 ó 60 cobayas sospechosos de peste, nos hizo desistir de este empeño. No hace mucho tiempo que uno de los miembros de la Comisión inglesa (el profesor Petri) decía a uno de nosotros que tal procedimiento riguroso no era necesario, y nos contaba que durante su permanencia en la India las ratas pestosas eran reconocidas como tales por el simple examen macroscópico, sin necesidad de ulteriores exámenes, cuando ya se tenía mucha práctica en ello, en prueba de lo cual, esta investigación la practicaban en la India durante aquella campaña los soldados indígenas aleccionados para esto.

Nosotros, respetando esta opinión, por proceder de una de las más altas y justamente reconocidas autoridades en la materia, insistimos en nuestra afirmación anterior, confesando que a nuestra estadística de 4.268 ratas examinadas por el procedimiento generalmente empleado preferiríamos la de 300 únicamente, por ejemplo, pero estudiadas por la inoculación directa de sus vísceras sin examen bacteriológico previo. Claro está que el procedimiento de examen macroscópico puede bastar en aquellas zonas pestosas donde quizás la epizootia pueda alcanzar grandes proporciones, ero no así donde se desarrolle una epizootia discreta.

b) HEPATICOLA HEPATICA.—En las páginas anteriores hacemos referencia al *Hepaticola hepatica,* nematode de la familia de los Tricuridos, que pone sus huevos en pleno tejido hepático, produciendo unas lesiones cirróticas que pueden inducir a error con la peste, si bien, vistas ya unas veces, se distinguen perfectamente. Como anteriormente decimos, todos los exámenes que se practiquen tomando estas lesiones por bacterianas y con miras a la búsqueda del germen causal serán negativas (frotis, cultivos e inoculaciones), y únicamente los cortes histológicos darán el diag-

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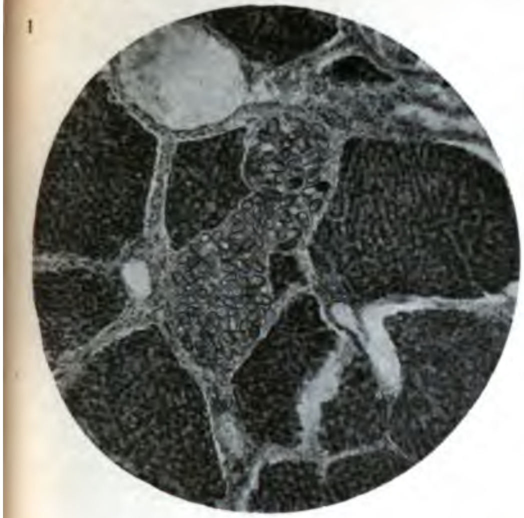

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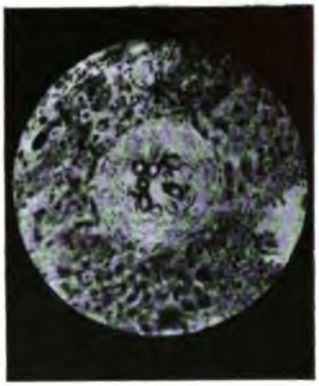

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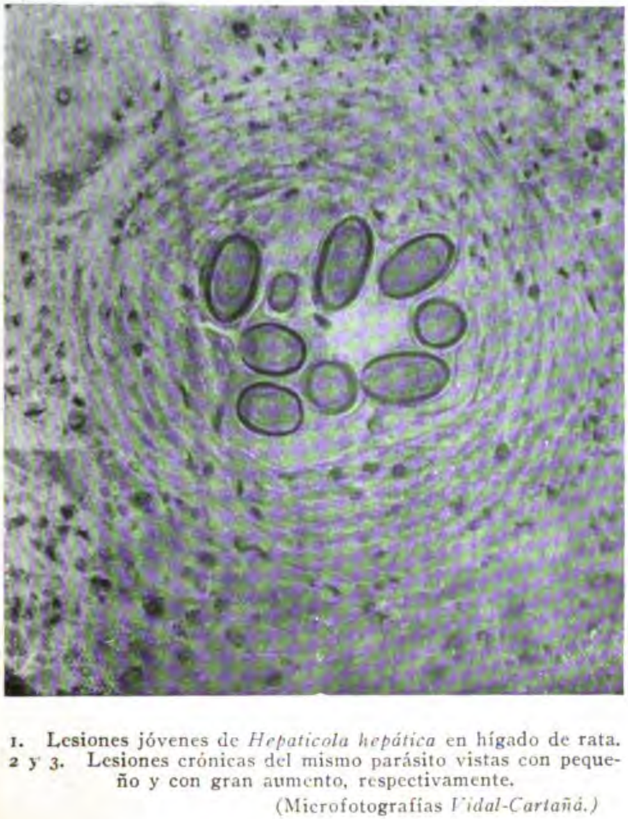

1. **Lesiones** jóvenes de *Hepaticola hepática* en hígado de rata.
2 y 3. **Lesiones** crónicas del mismo parásito vistas con pequeño y con gran aumento, respectivamente.

(Microfotografías *Vidal-Cartañá.)*

nóstico por observación en ellos de los huevos del parásito y las lesiones producidas, que pueden verse claramente en las microfotografías adjuntas. Estas observaciones pudimos hacerlas fácilmente (el Dr. Balen y Cartañá) durante la primera parte de este trabajo (julio de 1931), que precedió al que exponemos ahora por el hecho de que el primer lote de ratas estudiado pertenecía a un foco estrictamente limitado (cuartel viejo y deshabitado de la Barceloneta), donde casi todas las ratas (el 88 por 100) estaban parasitadas por este nematode.

Los huevos de este trichurido son muy parecidos a los del tricocéfalo, si bien son más alargados. La infección tiene lugar por vía digestiva; los huevos ingeridos liberan las larvas al llegar al intestino, las cuales llegan al hígado, donde terminan todo su desarrollo completo, verificando las hembras la puesta de los huevos en el mismo tejido hepático. Dada la imposibilidad de la salida de los huevos al exterior para perpetuar la especie, es probable que ésta tendrá lugar gracias al canibalismo de las ratas, que se infectarán devorando a sus congéneres parasitados.

c) QUISTE PRODUCIDO POR LA LARVA DE LA "TAENIA CRASSICOLIS" *(Cisticercus fasciolaris).*—Durante el mismo período a que acabamos de referirnos (julio de 1931) vimos con el Dr. Balen frecuentemente en el hígado de las ratas esta formación quística producida por el cisticerco de un cestode, observación que hemos tenido ocasión de hacer frecuentemente después durante el curso de nuestro trabajo desde noviembre de 1931.

Se trata de una tenia cuyo cisticerco llegado al hígado comienza su desarrollo dentro de una cavidad formada a expensas del tejido donde se aloja, y sin esperar a llegar al intestino de un segundo huésped empieza su desarrollo dando anillos numerosos que, si bien no llegan a maturación (indiferenciados), le dan el aspecto de un gusano adulto; la longitud de esta larva es muy variable de unos ejemplares a otros, según hemos tenido ocasión de observar, y depende del momento en que se encuentre de su crecimiento, y por lo tanto, el número de sus anillos, que puede ser de muy pocos: 10, 20 a 50, y muchos más; en la mayor parte se observa la formación esférica o bolsa (cisticerco), de donde parten todos los anillos.

El adulto es parásito del intestino delgado del gato, mide de 20 a 30 centímetros y tiene un rostro armado. Este cisticerco produce a veces un sarcoma en el hígado de las ratas parasitadas, si bien, como se desprende de los recientes estudios de Larrousse (1932), la ingestión de huevos de *Taenia crassicollis* va siempre seguida de cisticercos en el hígado, pero no siempre de producción de sarcomas.

El mismo autor citado y Borrel, en estudios anteriores (1930), concluyen que este cisticerco puede servir de vector para el bacilo de la lepra murina.

d) LESIONES SEUDOTUBERCULOSAS PRODUCIDAS POR EL "CORYNEBACTERIUM KUTSCHERI".—Nos referimos a los nódulos, que hemos tenido ocasión de observar dos veces en el tejido pulmonar de las ratas, producidos por este germen difteroide, grampositivo, llamado *Corynebachterium kutscheri,* que mencionamos ya en páginas anteriores. Esta infección y el germen que le produce nada tiene que ver con la seudotuberculosis *rodentium* o *pseudopestis,* que hemos estudiado antes.

e) ESPIROQUETOSIS ICTEROHEMORRÁGICA.—Esta investigación la hemos llevado a cabo inoculando a cobayas con la emulsión obtenida del producto de trituración estéril de los riñones y cápsulas suprarrenales de las ratas. Hemos inoculado de dos a cuatro cobayas por cada lote de ratas de número indeterminado. La vía seguida ha sido la intraperitoneal, y la cantidad de emulsión inoculada, de 2 a 5 cc.

Los resultados han sido positivos desde el primer momento en todas las ratas procedentes de Barcelona, especie *Epimys Norvegicus,* y en las dos terceras partes de las procedentes del Delta del Ebro.

A partir de los primeros resultados positivos, hemos entretenido el virus, haciendo pases por cobayas, y cuyos detalles resumiremos brevemente.

Curso clínico de la infección en los cobayas.

La inoculación intraperitoneal de 2 a 5 cc. de emulsión obtenida con el triturado estéril de riñones y cápsulas suprarrenales de las ratas en estudio o de cobayas ya espiroquetósicos (hígado, riñón, cápsula, ganglios, etcétera), nos ha producido el siguiente cuadro de infección:

Período de incubación, de cinco a seis días, durante el que ofrecen solamente ligero enflaquecimiento con temperatura normal. A partir del sexto o séptimo día, fiebre de 40 a 41°, enflaquecimiento, flacidez muscular, pelo erizado, etc. Del séptimo al octavo día, ictericia (ostensible en hocico, orejas, conjuntivas, patas, mucosa genital, etc.). Después, casi al mismo tiempo o al día siguiente, hipotermia, que precede de muy poco a la muerte. *(Crisis nefasta,* de Sanarelli.)

Durante una serie de pases, la duración ha sido casi siempre de unos ocho días, manteniéndose así desde el primero al último pase. Sin embargo, en una ocasión nos pareció observar un aumento en la virulencia, puesto que los cobayas morían en un plazo que se iba acortando en cada pase hasta llegar a constituir el cuadro típico que hemos descrito, en cuatro días. Los cobayas ofrecían al mismo tiempo un cierto piqueteado en el hígado. El hecho era debido a la asociación de una infección por colibacilo. Los cobayas del paso siguiente murieron ya antes de las cuarenta y ocho horas, con un cuadro de autopsia por entero a esta infección colibacilar. A este propósito recordamos lo que dice Sanarelli en sus estudios sobre la patogenia de las leptospirosis, que nosotros hemos leído posteriormente a la cnstatación citada de que la muerte en ellas es siempre debida a la asociación de ciertos gérmenes de salida (colobacilo, estrepto, paratíficos, etc.).

Después de la inoculación, hemos tomado diariamente la temperatura rectal de los cavias. A continuación detallamos las temperaturas de algunos de los primeros observados:

	DIAS								
	1.°	2.°	3.°	4.°	5.°	6.°	7.°	8.°	9.°	
Cobaya A...	38,5	39	38,8	39,2	39,5	40	*37,3*	Muerte al 7.° día.		
Cobaya B...	39,1	39,3	39,2	39	39,1	39,3	40,7	40,6	*37,4*	Muerte al 9.° día.
Cobaya C...	38,6	38,8	39	38,9	39,7	39,5	40,5	40,7	*37,2*	Muerte al 9.° día.

Resultados de autopsia.

Intensa ictericia observable en todos los tejidos; pero, sobre todo, en el tejido hipodérmico, intensa congestión hemorrágica en los cuatro grupos ganglionares axilares e inguinales. Congestión hemorrágica en el intestino grueso. Congestión hemorrágica en las cápsulas suprarrenales. Congestión hemorrágica en los órganos de reproducción en las hembras. Típicos focos hemorrágicos en pulmón perfectamente delimitados. Los cobayas machos, inoculados con ratas del Delta, presentaban además orquitis.

Sobrevivencia de algunos cobayas a la infección.

En las mismas condiciones de inoculación, no todos los cobayas mueren. Algunos de ellos siguen el mismo curso de la infección (incubación, enflaquecimiento, erizamiento del pelo, flaccidez muscular, fiebre e intensa ictericia), pero consiguen salvarse.

Inmunidad de los cobayas que han sufrido una primera infección.

Los cobayas que han sobrevivido a una primera inoculación han resistido una segunda. Ejemplo típico nuestro, C. número 2-39, que habiendo sufrido la infección del 23 al 30 de enero por inoculación de 2 cc. de emulsión, resiste el día 15 de marzo una *inoculación de 10 cc.,* mientras que mueren con el cuadro típico y el período corriente dos cobayas testigos.

Prueba de la eliminación del virus por la orina de los cobayas infectados.

Con el fin de ver hasta cuándo eliminaban espiroquetes por la orina los cobayas curados y, por lo tanto, hasta qué punto podían constituir un reservorio de virus, inoculamos la orina extraída por sondaje a cobayas nuevos. Los resultados no han sido demostrativos, si bien algunos de estos cavias morían en plazo largo, con gran congestión de las suprarrenales y focos hemorrágicos pulmonares.

Ensayos de transmisión con ácaros.

Esta prueba la hemos realizado con ratas blancas, inoculadas por vía intraperitoneal con emulsión de virus espiroquetósico controlado. Los resultados han sido negativos. De todos modos, esta prueba nos ha sido útil para demostrar la ***resistencia de la rata blanca a esta infección.*** Ha resistido perfectamente la inoculación, pero ***ha conservado el virus durante todo el tiempo,*** puesto que dos meses después de aquélla hemos inoculado con el triturado de sus vísceras a cobayas nuevos, que han muerto con el cuadro típico de espiroquetosis.

Cultivos.

Utilizando el medio de Pettit, suero de conejo diluído al 5° en suero fisiológico, y sembrando riñón, hígado o sangre, no hemos podido obtener resultados positivos hasta ahora.

Cortes histológicos.

Nitratación procedimiento Cajal: han demostrado la presencia de espiroquetes en pulmón y en cápsula suprarrenal; pero, sobre todo, muy abundantes en riñón. (Véanse los adjuntos dibujos.)

Encuesta en el hombre.

La existencia de la espiroquetosis en las ratas capturadas en el Delta del Ebro nos hizo llevar la encuesta en el hombre, ya que el tipo del trabajo obliga al natural del país (cultivo arrozal) al frecuente contacto con las aguas, que pueden estar polucionadas con los excretas de las ratas. La idea de la posible existencia de esta infección en nuestra zona había sido sugerida, hace ya muchos años, por el Dr. Vilá, de Tortosa, uno de los primeros y más distinguidos discípulos y colaboradores de la escuela de Pittaluga en Cataluña.

La encuesta, que comenzamos en 1931 junto con el Dr. Torné, de San Carlos de la Rápita, nos permitió ver dos casos, cuyas historias clínicas a continuación detallamos:

Caso primero.—A. R., diecinueve años, cultivador de arroz; antece-

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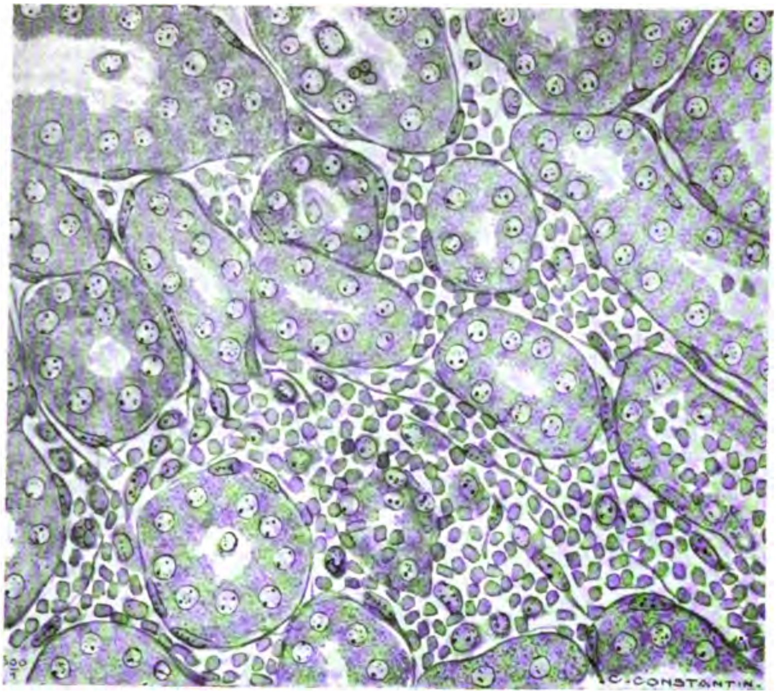

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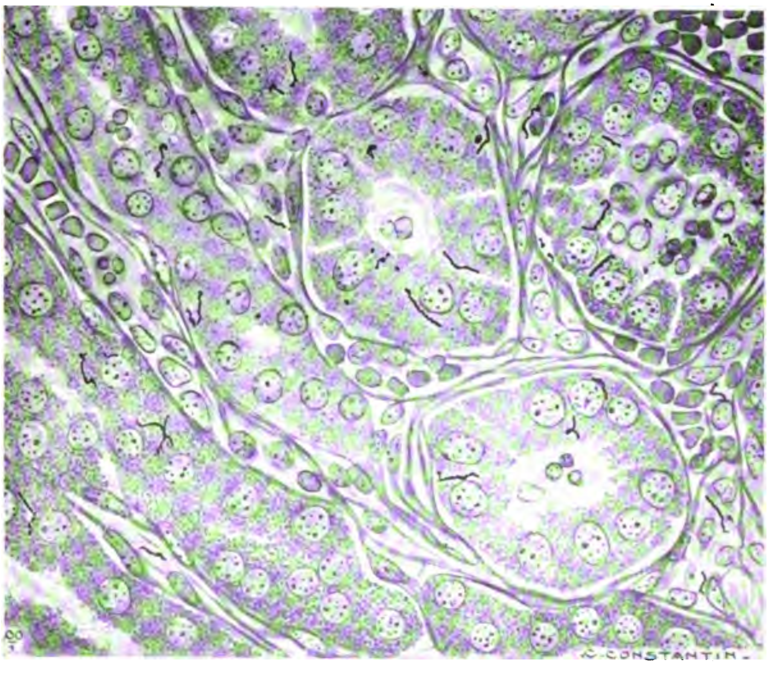

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Cortes histológicos del riñón de cobaya, teñidos por el método de nitratación de Cajal, en donde aparecen típicamente los espiroquetes. 1 × 600, 2 × 800 diámetros.

(Dibujo de *Constantin.)*

dentes personales y familiares sin importancia. Enfermedad actual comienza el 20 de junio, con cefalalgia.

El 21 guarda cama; temperatura, 38°, y por la tarde, 38,5°. Pulso, 100. Cefalalgia, postración, náuseas, lengua saburral, estreñimiento, angina eritematosa, ligera rigidez nuca fotofobia, reacción pupilar normal.

El día 22, el mismo cuadro, más intenso. Voz velada, esputos faríngeos *sanguinolentos*. Límite superior, matidez hepática cuarto espacio intercostal.

Día 23: temperatura, 38,6° y 104 pulsaciones. Aumenta rigidez nuca. *Epistaxis abundante.*

Día 24: temperatura mañana, 37,8°; tarde, 38,7°. Pulsaciones, 100, oliguria. Orina oscura, indicios albúmina. Kernig ligero. Intensa rigidez nuca. Reflejos rotulianos disminuídos.

Día 26: temperatura, 38,6°; pulsaciones, 115. *Nueva epistaxis* abundante. Obnubilación intensa, casi coma. Rigidez nuca y Kernig notables. Punción lumbar, líquido normal. Hemocultivo y serodiagnóstico, negativos por dos veces.

Día 27. Mejoría. Temperatura, 28°; pulsaciones, 90. Disminuye todo el cuadro. Oliguria. Orina negra. *Indicio albúmina.*

Día 28. Mejoría. Temperatura, 36,6°; pulsaciones, 60. *Esputos sanguinolentos*. Adenitis angulomaxilar derecha, con desarrollo rápido e intensa periadenitis. *Noche: temperatura, 40°; pulsaciones, 90;* pero se siente algo mejor que los primeros días. Sensorio claro. Persiste algo rigidez nuca. *Ictericia intensa*. Orina oscura. Indicios pigmento, sales biliares.

Del 29 al 2, disminuye la temperatura, a partir del cual persiste la apirexia. Durante estos días ha continuado la ictericia.

Entre el día 2 y 3 hemos inoculado siete cobayas, dos con sangre y cinco con orina centrifugada de diferentes micciones. Tres de estos últimos han muerto a los ocho y nueve días, con el cuadro típico de la espiroquetosis.

CASO SEGUNDO.—E. M., treinta y ocho años, pastor. Enfermedad actual comienza el 19 de agosto, *con dolores en las pantorrillas* y cefalea intensa.

Día 21. Cefalalgia, vómitos, *dolores de pantorrillas;* temperatura, 38,5. Día 22. Apirético por la mañana. Tarde, cefalalgia, vómitos, dolores en pantorrillas y miembros. Temperatura, 38,5.

Días 23, 24 y 25. El mismo cuadro. Algún *esputo hemopótico.*

Día 26. Ictericia. *Indicios de albúmina* en orina.

Día 27 y siguientes se acentúa la ictericia y llega a ser intensísima. Se inoculan tres cobayas con orina centrifugada de diferentes micciones, y uno de ellos muere a los diez días, con cuadro típico de espiroquetosis.

Sigue mejorando desde el 29 en adelante. Sin recaída.

Tratamiento.—Sintomático y cyclotropina intravenosa.

Posteriormente (verano de 1932) hemos encontrado *otro foco de cinco* *enfermos* trabajadores, también del cultivo de arroz y habitantes de la partida de Jesús y María, en el Delta izquierdo del Ebro; dos de ellos habían presentado *hematurias*.

Durante este mismo tiempo (verano de 1932) contribuímos al diagnóstico de *un pequeño foco en Prat de Llobregat* (Barcelona), que ha tenido lugar entre obreros empleados en la limpieza de unas acequias de regadío. Vimos los enfermos con el Dr. Figueras, médico de la localidad, que los había observado y diagnosticado clínicamente, habiéndolos notificado luego a la autoridad sanitaria. Dichos enfermos, en número de cuatro, habían presentado un cuadro clínico de ictericia espiroquetósica, y la inoculación de centrifugado de orina al cavia dió resultado positivo.

Ultimamente, durante los meses de agosto y septiembre de 1933, *han sido observados 11 casos en Barcelona* por los Dres. Pedro Pons y J. Surós (*Revista Médica*, de Barcelona, octubre 1933), que fueron estudiados y diagnosticados clínica y bacteriológicamente en el servicio del profesor Pedro Pons, de la Facultad de Medicina; las pruebas de laboratorio demostraron la presencia del Leptospira icterohemorrágica en el centrifugado de la orina de los enfermos, y además, la inoculación al cobaya fué positiva. El contagio fué en casi todos por vía digestiva, y en dos de ellos pudo incriminarse los baños tomados en aguas polucionadas por las deyecciones de las ratas. La letalidad fué del 27 por 100 (tres defunciones en los 11 casos). La circunstancia de que casi todos esos enfermos habían bebido agua de la misma fuente poco antes de enfermar, hizo sacar la deducción epidemiológica al profesor P. Pons de que pudiesen encontrarse contaminadas aquellas aguas, comunicándolo así a las autoridades sanitarias, que encargaron a uno de nosotros (Cartañá) de la verificación de dicha encuesta, cuyo resultado fué la comprobación exacta, puesto que encontramos ratas portadoras del leptospira cazadas en los alrededores de dicha fuente, y además, la demostración directa del leptospira en dicha agua. La investigación fué practicada en la fuente del M. G., situada en la montaña de Las Planas, sitio cercano a Barcelona, donde concurre la población en gran número los días festivos, y por lo cual quedan desperdicios alimenticios que atraen gran cantidad de ratas. La constitución del terreno, pizarrosa, permite fácilmente la polución de las aguas subterráneas por las deyecciones de las ratas, y por otra parte, el tránsito rápido, condición que favorece dicha contaminación, impidiendo que pueda morir el leptospira antes de llegar al agua, ya que la resistencia del leptospira en la orina eliminada es escasa (veinticuatro horas), siendo en cambio su vitalidad en las aguas mucho más larga.

La técnica que seguimos para el aislamiento del virus fué la siguiente: Recogimos dos muestras de agua de 20 litros cada una, sometiéndolas para la concentración de gérmenes: una, a la precipitación por el hidrato de alumina coloidal, y a la otra, a filtración por bujía Chamberland L. 2. El precipitado fué inoculado en cantidad de 5 cc. por vía intraperitoneal, a dos cobayas, y el producto del lavado, con solución salina y raspado de las paredes de la bujía filtrante, a dos cobayas más; la primera prueba dió resultado positivo, muriendo uno de los dos cobayas con el cuadro típico de la espiroquetosis icterohemorrágica, a los ocho días, y demostración del leptospira en las vísceras afectas. Los cobayas inoculados con el producto del lavado de la bujía filtrante sobrevivieron, no presentando síntoma ninguno. Este hecho está perfectamente de acuerdo con la característica de filtrabilidad que posee el leptospira icterohemorrágico, como ha sido demostrado por Inada, Ido, Hübener y Reiter, cualidad que, por otra parte, ha sido utilizada por estos investigadores para obtener el leptospira en el cultivo puro, partiendo de cultivos impurificados por bacterias ya que la filtración permite el paso del leptospira y retiene las bacterias del cultivo.

Así, pues, no cabe ninguna duda de que dicha epidemia mencionada fué de *origen hídrico,* tal como, por ejemplo, la que sucedió en Lisboa en agosto y septiembre de 1931, descrita por el profesor Ricardo Jorge.

Anteriormente a todos estos casos citados por nosotros en el Delta del Ebro (San Carlos de la Rápita, San Jaime), Prat de Llobregat y los de Barcelona, por el profesor P. Pons, han sido descritos en España dos casos probables por el Dr. Vilá, de Tortosa, en el año 1916; un caso por Urtubey y dos casos por el profesor Manuel Tapia, de Madrid, en Albacete. El profesor Pedro Pons llama la atención sobre una serie de casos de ictericia infecciosa que se observaron en Barcelona, precisamente este verano pasado, y de los que, aunque no se haya tenido certeza diagnóstica por la falta de diagnóstico bacteriológico, cabe la sospecha de un probable origen espiroquetósico.

Por esta razón creemos que esta infección es más extendida en España de lo que anteriormente se creía, y debería en este sentido investigarse el espiroquete de Inada e Ido en todos los casos de ictericia sospechosa, y máxime cuando su benignidad en algunos casos y la falta de un síndrome característico del cuadro de la infección espiroquética pueden hacer incluir muchos enfermos en otros grupos de ictericia.

CAPITULO VIII

LA PESTE BUBONICA EN BARCELONA

La peste se ha manifestado en Barcelona, durante el presente siglo, en diversas ocasiones, si bien con poca intensidad, en focos limitados y de corta duración.

A continuación expondremos un resùmen de los diversos brotes epidémicos o de los casos aislados que se han observado.

La distribución topográfica puede observarse fácilmente en el plano que acompañamos, donde hemos situado todos los focos desde el año 1905.

La primera epidemia de peste en Barcelona (en los tiempos modernos) tuvo lugar el año 1905. Duró del 19 de junio de 1905 al 20 de abril de 1906, y hubo 52 casos positivos y 15 probables.

Los focos principales se desarrollaron desde el Paralelo hasta Casa Antúnez (Sudeste de la ciudad), y se manifestaron alrededor de los estercoleros y de los depósitos de basuras domiciliarias.

1905	Junio	2	casos.
—	Julio	15	—
—	Agosto	2	—
—	Octubre	2	—
—	Noviembre	6	—
—	Diciembre	2	—
1906	Enero	19	—
—	Marzo	3	—
—	Abril	1	—

La marcha fué la siguiente: Comenzó en 19 de junio, siguieron luego 15 casos en julio y dos en los primeros días de agosto. ***Siguen luego dos meses sin ningún enfermo nuevo,*** apareciendo otra vez dos casos en octubre, seis en noviembre y dos en diciembre. Aumentan de nuevo en enero (19 casos), ***siguiendo otra pausa de cuarenta y tres días,*** para presentarse tres nuevos casos en los primeros días de marzo; ***sigue luego otra pausa de cuarenta y ocho días sin ningún caso*** (4 de marzo a 20 de abril). y la última invasión, en 20 de abril de 1906.

Origen murino: Importanción indudable, puesto que *no se conocía la peste en Barcelona desde 1681.*

La mortalidad fué de 19,60 por 100.

La de los asistidos *en el hospital, solamente* de 11,11 por 100.

Año 1919.

En este año (trece años después de la anterior epidemia) hubo nueve casos de peste bubónica. El foco fué un almacén de depósito de granos procedentes de la Argentina, situado en la avenida Icaria (almacenes Xelma); seis o siete de los casos eran trabajadores de dichos almacenes. Origen probable, ratas infectadas o solamente pulgas que vinieron con los cargamentos de trigo.

Los almacenes estaban en buenas condiciones, y la epizootia no se propagó.

Este brote tuvo lugar en el mes de septiembre, y un caso en los primeros días de octubre.

Año 1920.

Se dió un caso de peste a bordo del vapor *Condesa Adelma,* que procedía de las costas de Italia, y que llegó a Barcelona el día 8 de julio. Por orden de la Dirección de Sanidad Exterior, fué enviado al Lazareto de Mahón; pero, además, se hizo activa campaña de desratización en la zona del puerto y se vigiló rigurosamente durante doce días a los trabajadores que habían intervenido en la descarga del citado vapor, sin que ninguno de ellos presentara novedad. El médico alemán Dr. Kellner, que había visitado al enfermo pestoso a bordo del vapor, sufrió, al cabo de tres días, una adenitis axilar y un bubón inguinal con fiebre, pero sin gran gravedad, cuadro sintomático que duró unos ocho o diez días, curando completamente después. Este médico había recibido, el mismo día que visitó al enfermo del barco, una inyección de suero antipestoso de 20 cc.; se interpretó pues, como un caso de infección pestosa atenuada por la acción del suero. Se aislaron y vacunaron todos los vecinos de la misma casa, sin que ocurriera luego novedad alguna.

Año 1922.

Hubo unos 28 casos. Los focos principales fueron una fábrica de harina, La Gallarda, situada en la carretera de Ribas (Nordeste de la ciudad), y la estación del Norte; otros casos, en el paseo del Cementerio, calle Wad-Ras y muelle.

No hubo participación en los grandes focos murinos (Hospitalet, San Martín, Casa Antúnez), ni aun se notó participación de las ratas de los sitios donde se desarrolló el brote.

Hubo dos casos en trabajadores del muelle: uno, trabajador del muelle de carbón, y otro, del tinglado número 7 (tráfico de algarrobas), que enfermó días después de haber arrojado al mar unas ratas que encontró muertas en aquel departamento (según declaración del enfermo).

Esta epidemia estuvo, pues, localizada en la parte Este y Norte de la ciudad, aunque no puede verse relación de estos dos últimos casos con los focos anteriores.

La zona peligrosa, en la que se intensificó la desratización, fué la comprendida en los límites siguientes: Puerto hasta Pueblo Nuevo; Dos de Mayo, Wad-Ras, Marina, carretera de Rivas, Comercio hasta el mar, Pero el número de ratas que se encontró fué reducidísimo, según dicen los que dirigieron aquella campaña. Esta epidemia dió comienzo el 3 de septiembre y terminó el 24 de noviembre.

Octubre	16 casos.
Septiembre	7 —
Noviembre	5 —

Año 1923.

Primer caso: 24 de octubre. Calle Aguilar, 12. En el patio-corral de la casa, el enfermo había encontrado días antes cuatro ratas muertas y las había arrojado a la alcantarilla. El número de ratas había aumentado desde que se habían removido los trapos de una trapería cercana.

Segundo caso: 6 de noviembre. Empleado en la selección de basuras de la calle del Comercio, barriada de Santa Eulalia (Hospitalet), vivía en la calle de San Erasmo. Según indicación del enfermo, había ratas en cantidad enorme, habiéndose encontrado muchas de ellas durante aquel tiempo.

Año 1925.

Día 4 de marzo. Un caso (calle de Lauder, número 1) en un comerciante de plátanos, que había recibido un cargamento procedente de las islas Canarias. En el embalaje se encontró una rata, cuyo examen bacteriológico demostró la infección pestosa.

Año 1930.

Primer caso: 30 de octubre. Calle de Roger, 283. Obrero de una fábrica de vidrios de Hospitalet.

Ningún resultado dió la encuesta epidemiológica en dicha fábrica ni en la casa donde habitaba el enfermo. En cambio, siguiendo las investigaciones, se encontró que las tres personas que constituían la familia de la esposa del enfermo, que habitan en la calle-carretera de la Bordeta, número 56, habían muerto de enfermedad muy rápida en pocos días de intervalo; la madre había muerto el día 8 de octubre, de un modo inesperado y rapidísimo (diagnóstico, bronconeumonía); a los pocos días enfermó su esposo, que moría el día 21 de octubre, con el diagnóstico de cálculo nefrítico, y el 22 de octubre fallece la abuela, con el diagnóstico de gastroenteritis. Estudiados posteriormente, con los médicos de cabecera, los síntomas que presentaron estos enfermos, se dedujo que se trataba de peste; por lo tanto, que el foco de este pequeño brote se encontró allí, carretera de la Bordeta, 56. Además, se constató la abundancia de ratas en esta casa y alrededores.

embre de 1931

Casos	Mes	Fuente probable	Terminación
1	Agosto	Desconocida.	Fallecida.
2	—	Almacén trapos "Pantinat".	Curación.
3	—	Basureros y trapos C. Aprestadora.	Idem.
4	—	Idem C. Paretos, 67.	Fallecida.
5	—	Idem C. Independencia, 26.	Idem.
6	—	Idem íd. o contagio familiar.	Curación.
7	—	Idem.	Idem.
8	—	Trapería C. Bofarull, 235.	Fallecido.
9	—	Basurero frente su vivienda.	Curación.
10	—	Trapería vecina.	Idem.
11	—	Basurero Hospitalet "Coloma".	Idem.
12	—	Basurero y trapería Torrent Guinéu, 53.	Idem.
13	—	Idem íd. íd.	Fallecida.
14	—	Idem íd. íd.	Curación.
15	—	Idem íd. íd.	Idem.
16	Septiembre	Basureros vec. o fáb. cartón Casanovas	Idem.
17	—	Idem C. Aprestadora.	Fallecida.
18	—	Almacenaba su casa desechos matadero.	Fallecido.
19	—	Basurero C. Igualdad.	Curación.
20	Octubre	Desconocida.	Idem.
21	—	Basureros Club Marina.	Fallecido.
22	—	Idem íd. íd.	Curación.
23	—	Idem íd. íd.	Idem.
24	—	Desconocida.	Idem.
25	—	Conviviente caso anterior.	Idem.
26	Noviembre	Basureros y trapos vecinos.	Idem.

CAPITULO IX

EPIDEMIA DE 1931

El brote epidémico de 1931 se desarrolló del 5 de agosto al 12 de noviembre, con un total de 26 casos, en zonas de los alrededores de Barcelona no urbanizadas.

El foco *de mayor importancia fué en Hospitalet, con 17 casos;* otro foco de menos importancia, y muy limitado (cinco casos), en Torrent de la Guineu, Noroeste de la ciudad, y además, cuatro casos dispersos en una zona extensa cercana a Hospitalet (Sans, carretera de la Bordeta, carretera del Puerto y Casa Antúnez), que podemos considerar como otro foco. En resumen, la mayor parte de los casos se dieron en la parte Sur de Barcelona, y un foco de extensión limitada y de cinco casos, en otro sitio muy distante, situado en la parte Noroeste.

Relación con basuras y trapos.—Las tres zonas tienen como carácter común la existencia de los depósitos de basuras domiciliarias de la capital, basuras que, como decimos después, constituyen los núcleos más importantes de la población murina.

Casi la totalidad de los atacados fueron individuos en inmediato contacto con estas *basuras* (empleados en la selección y aprovechamiento), vecinos de estos depósitos o en relación con *trapos viejos* (almacenes de trapos viejos, que por su relación comercial con la selección de basura están situados en las mismas zonas).

Los casos se sucedieron en el tiempo en el orden que indicamos en el cuadro resumen de enfermos y en la gráfica adjunta.

Falta de contagio interhumano.—Salvo en un caso (niña de ocho meses, hermana del caso número 5), no se ha visto contagio interhumano; de otra manera, hay una falta absoluta de relación, de enlace, de unos enfermos con otros, no existiendo una "cadena de casos de peste", como fácilmente se deduce al estudiar el conjunto de este brote en relación con el tiempo de su aparición de cada caso y su distribución topográfica.

Los casos que pudieran ofrecer una duda respecto a los dichos, por haberse presentado varios en una misma casa, son los dos grupos de enfermos del "Torrent de la Guineu" y de la "Masía de ca'l Tocayo". En el primer grupo se trataba de un pequeño taller de curtidos de pieles de conejos, situados a planta baja y dentro de un cercado que servía de almacén de trapos viejos y en relación con un depósito de basuras contiguo. Hubo cuatro casos, y comenzó la enfermedad el mismo día o con un día de diferencia.

En la "Masía de ca'l Tocayo", los hechos se desarrollaron de la siguiente manera: Dicha masía es una casa aislada, y la más cercana, a un núcleo separado de basureros, compuesto de 110 depósitos de basuras en las peores condiciones higiénicas imaginables, sin ninguna casa de fábrica (entonces), y donde pululan en cantidad asombrosa las ratas. Las lluvias copiosas acaecidas entre el 10 y el 17 de octubre anegaron completamente aquellos montones de basuras y criaderos de ratas, y éstas y las pulgas, sobre todo estas últimas, se refugiaron en el único edificio que tenían más cercano ("Masía de ca'l Tocayo"). La familia que lo habita vióse sorprendida por esta invasión repentina de pulgas de tal manera, que, según nos contaron, no se podía bajar a la planta baja de la casa y a la parte de la era no inundada sin ser fuertemente picado por un gran número de ellas; además, vieron alguna rata enferma en pleno día. A los cuatro días presentró los primeros síntomas de peste uno de los familiares (el padre), que murió a los seis días, y luego enferman el mismo día los tres hijos. Es de notar, sin embargo, el hecho de que en el foco de basureros próximos no se dió ningún caso de peste conocido.

Aunque de estos dos grupos de enfermos que hemos citado, los individuos atacados hayan sido todos de una misma familia, o convivientes, tampoco creemos que pueda verse en ellos un contagio interhumano, o sea una relación de dependencia de un caso con otro, por las dos consideraciones de que era evidente la existencia de un factor externo común a todos ellos (ratas y pulgas), y además, porque se produjeron casi todos los casos al mismo tiempo. Por lo tanto, creemos mejor, con el doctor Claramunt, aplicarles el calificativo de "contagios colectivos".

Evolución de la epidemia.

Como vemos en el gráfico adjunto (fig. 6), comenzó con un caso el día 5 de agosto, siguió con una *pausa de catorce días* sin ninguna invasión, siguen 15 casos desde el 19 al 28 de agosto.

Nueva pausa de trece días, desde el 19 de agosto al 11 de septiembre, a la que siguen cuatro casos, del día 11 al 23 de septiembre (mediando una semana en los dos últimos).

Otra pequeña pausa de siete días, del 23 de septiembre al 1.° de octubre, día en que hubo un solo caso nuevo.

Sigue *otra nueva calma de veintitrés días* (del 1.° de octubre al 24), seguida de cuatro casos de un solo día (día 24) y otro caso al cabo de cinco días.

Nueva pausa de trece días (29 de octubre a 12 de noviembre) y último caso en 12 de noviembre.

Características epidemiológicas.—En este gráfico se objetivan claramente las características de este brote en cuanto al tiempo de aparición de las sucesivas invasiones. Mediaron catorce días entre el primer caso y el brote más importante; este brote, aunque de una mayor densidad que el restante número de casos, tampoco tiene el carácter de *explosión,* puesto que necesitó nueve días para producir 15 casos. Después de ellos, empieza una *marcha francamente intermitente,* siendo la *declinación muy larga,* con aparición de nuevos casos después de *calmas engañosas.*

Otra característica muy importante, que aunque no se evidencia en este gráfico se observa claramente en el mapa adjunto, es la de la distribución de los casos que demuestran que, al propio tiempo, hubo *discontinuidad* o *dispersión topográfica,* o sea que no sólo se desarrollaron en casas y calles que no tenían relación unas con otras, sino en barrios y zonas distintos y alejados, algunos de ellos muy distantes, tales como Hospitalet y Torrent de la Guineu, Sur y Norte de la ciudad, respectivamente, o Casa Antúnez, carretera del Puerto, etc. En conjunto, hubo 15 casos en agosto, cinco en septiembre, cinco en octubre y uno en noviembre.

Origen murino de la epidemia de 1931.

El estudio de esta epidemia conduce a la conclusión de que fué precedida de una epizootia o concomitante con ella; de otra manera, *el brote epidémico de 1931 tiene todas las características de una epidemia de origen murino.*

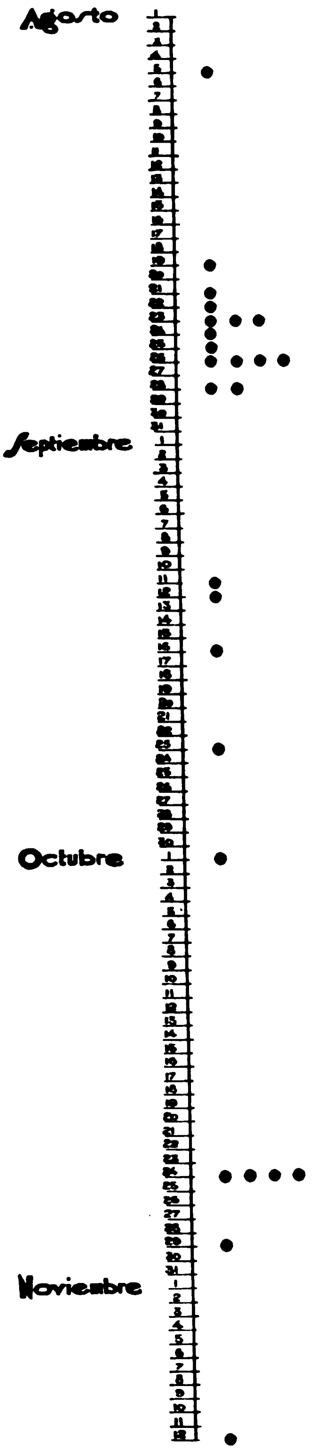


Fig. 6.—Distribución en el tiempo, de los casos de peste ocurridos en 1931

Los hechos que la epidemiología da como característicos y propios de las epidemias de peste bubónica de origen murino son las siguientes:

a) Comienzo lento, indeciso, solapado.

b) Evolución irregular no uniforme.

c) Un estudio de conjunto muestra una serie de paroxismos y de calmas.

d) Se trata como de una serie de epidemias parciales que, ya simultánea o sucesivamente, evolucionan por su cuenta como si fuesen independientes unas de las otras.

e) La declinación se anuncia progresiva, pero irregular aún y sujeta a remisiones a veces engañosas, dejando a veces secuelas de casos aislados.

f) Repartición topográfica irregular de los casos dispuestos como al azar.

g) Tendencia a cantonarse bastante tiempo en la aglomeración urbana donde ha sido importada.

Así, pues, lo que domina es lentitud, irregularidad, capricho en su evolución, carácter bastante circunscrito sin gran tendencia a la difusión; extensión progresiva, pero desigual; retornos periódicos.

La sola observación del cuadro-resumen de la epidemia de 1931 y del plano donde están señalados los casos es suficiente para ver que se adapta a estos principios enumerados, y que, por lo tanto, debe considerarse como de origen murino. De no admitirlo así, tendría que demostrarse la relación de unos casos con otros, cosa que no existió.

Caracteres clínicos.

En todos los casos se trató de formas ganglionar o bubónica, siendo los demás síntomas los clásicos de la grave infección por el bacilo de Yersin, que no entraremos en describir.

Hubo algún caso leve (como el último) y los otros de mayor intensidad.

El delirio fué tan intenso en uno de los enfermos, y se manifestó como primer síntoma aparente, que fué internado por su familia en un asilo municipal para dementes, donde, al ser reconocido por los médicos y establecer el diagnóstico sospechoso de peste, fué trasladado inmediatamente al Hospital de Infecciosos, donde se comprobó dicho diagnóstico.

La adenitis fué inguinal en 24 casos (10 de ellos, bubón doble) y cervical en los otros dos (niños de ocho años y ocho meses, respectivamente), hecho, por lo demás, frecuente en los niños de poca edad.

En dos o tres casos se observó el carbunco pestoso.

En la mayor parte de los casos evolucionó el bubón sin supurar; pero en seis hubo supuración y abertura de bubón, y en éstos la fiebre persistió con oscilaciones y remitencias durante quince o veinte días. En dos de los casos hubo necrosis de los tegumentos; uno de ellos era una mujer embarazada, que curó siguiendo el embarazo su curso normal.

El primer diagnóstico bacteriológico fué practicado *post-mortem* por el doctor Domingo, y los demás, en el laboratorio del Hospital de Infecciosos (punción del bubón frotis y siembras en agar y caldo).

Tratamiento utilizado.—Suero antipestoso del Instituto Nacional de Higiene de Madrid y del Laboratorio Municipal de Barcelona, a grandes dosis, 60 a 150 cc. diarios, ya sea por vía endovenosa o subcutánea. Las inyecciones de grandes dosis de suero iban seguidas de reacción. Posteriormente, algunos enfermos presentaban eritemas y artralgias.

La mortalidad.—Fué del 20 por 100 en los enfermos tratados; tres enfermos que no ingresaron en el hospital y no recibieron tratamiento específico murieron (primer caso, denunciado en 5 de agosto, y los dos primeros casos de la familia de la "Masía de ca'l Tocayo", 24 de octubre).

De los tratados que murieron, en algunos, el tratamiento no pudo ser precoz, puesto que la denuncia no fué pronta.

Caso número 4. Ingresa el 22 y muere el 23.

Caso número 5. Ingresa el 22 (después de siete días de enfermedad) y muere el 24.

Caso número 21. Ingresa el 24 de octubre y muere el mismo día.

Aclaración que hacemos, puesto que está en favor de la sueroterapia, ya que cabe la posibilidad de que hubiera podido seguir un curso favorable en el caso de ser tratados precozmente.

En resumen, la peste se ha manifestado en Barcelona durante los años siguientes: 1905, 1906, 1919, 1920, 1922, 1923, 1925, 1930 y 1931.

Descontando los casos importados, o sea aquellos que no dependían en modo alguno, ni tenían relación alguna con las epidemias anteriores de Barcelona (casos de 1920 y de 1925), tenemos: 1905-6, 1919, 1922, 1923, 1930 y 1931.

El estudio de estos diversos brotes epidémicos merece atención, porque nos lleva a la siguiente pregunta, importante desde el punto de vista de la epidemiología de la peste en Barcelona, y cuya respuesta puede razonarse por consideraciones epidemiológicas, ya que no por afirmaciones categóricas del dominio de la bacteriología:

¿Existe relación de dependencia entre todos estos brotes epidémicos? O de otra manera: *¿Puede admitirse que haya existido desde el año 1905 una enzootia pestosa en la población murina que explique estas explosiones de peste humana que hemos indicado? ¿O ha sido importado el virus responsable de cada uno de estos brotes?*

Aunque es difícil contestar categóricamente a esta reflexión, creemos que puede admitirse la relación de unos brotes con otros (sobre la base de la permanencia de la epizootia), *cuando el período intermediario entre una epidemia y otra es corto* (de otoño a invierno siguiente, de otoño a verano u otoño siguiente, etc.), tal, por ejemplo, las de los años *1905 y 1906* (que debe considerarse como una sola epidemia), y las de los años *1922 y 1923,* aunque mediara casi un año (último caso de 1922, en 24 de noviembre, y primer caso de 1923, en 24 de octubre). Y las de *1930 y 1931,* con todo y tener que observar que en algunas de ellas (1922 y 1923) los focos de un año y de otro no tenían relación de contigüidad ninguna.

Pero en los brotes epidémicos muy distanciados, 1906 a 1919 y 1929 a 1930, no creemos que debamos admitir relación ninguna de dependencia ni aceptar una enzootia solapada que pudiese guardar el virus de una epidemia a otra. Las razones que nos llevan a esta convicción son de dos órdenes: la primera, un tanto empírica, pero que no deja de tener su valor, es la siguiente: Si lo que motive la peste de origen murino en el hombre son los contactos entre el hombre, rata y pulga, y habiendo sido durante todos estos años exactamente iguales las condiciones de lo que pudiéramos llamar ambiente pestoso, y exactamente iguales las ocasiones de contacto, puesto que en las zonas de ratas de los alrededores de B. existe una verdadera convivencia entre hombre, rata y pulga. ha de ser difícil, si no imposible, de explicar cómo hubieran podido pasar trece años y siete, respectivamente, de enzootia sin un solo caso de peste humana. No nos extendemos más sobre esta consideración, que podrá apoyarse aún con otros datos.

La otra razón se deriva de los conocimientos que actualmente se tienen respecto *a las causas de una posible desaparición espontánea de la epizootia,* que permite explicar cómo una localidad puede verse libre espontáneamente de la peste murina.

Casi todos estos estudios se deben a la "Comisión inglesa de la peste de la India", a cuyos trabajos o publicaciones hemos acudido para el estudio de estos factores, así como a las explicaciones que hemos podido obtener de uno de sus más destacados miembros, al Dr. Petrie, actualmente profesor del "Lister Institut", de Londres.

Ellos se refieren principalmente a la inmunidad contra la peste que adquieren las ratas en el curso de una epizootia.

La "Comisión Inglesa" demuestra que, en general, la resistencia (inmunidad) de las ratas a la peste es proporcional a la prevalencia de la enfermedad en aquella localidad en que habitan. Así un gran número de ratas de Bombay mostraron una alta inmunidad a la peste (controlada por infección por vía digestiva y por transmisión por pulgas), al paso que otras ratas de zonas donde no había habido peste epizoótica no tenían esta inmunidad.

La resistencia natural de las ratas a la peste es debida no solamente a la previa recepción de dosis subletales e inmunizantes de bacilo pestoso durante la epizootia, sino a la inmunidad heredada de la nueva generación engendrada por las ratas sobrevivientes a la epizootia. Esto lo demostró la Comisión por el hecho de que las ratas jóvenes de Madrás Bombay y Poona poseían una inmunidad del mismo grado que la media de las adultas del mismo sitio. Además, experimentalmente se comprobó este hecho, porque las crías obtenidas en el laboratorio partiendo de ratas libres cazadas en zonas pestosas demostraron la misma resistencia e inmunidad que sus progenitores.

Es probable, además, que el proceso de extinción de la epizootia entre las ratas no consista tan sólo en su inmunidad activa adquirida durante el período epizoótico y la subsecuente transmisión de la inmunidad a su prole, sino en gran parte también a la eliminación de los miembros o elementos más susceptibles.

Mc Coy observó que alrededor del 50 por 100 de las ratas adultas (la mayor parte, *E. Norvegicus*) de San Francisco eran inmunes.

Asimismo, Swellengrebel y Otten, en 1914, comprobaron la inmunidad de las ratas de las casas en Java, por medio de transmisión experimental, encontrándose un gran número (que llegaba al 36 por 100) de ratas inmunes. A igual conclusión llegó Spencer en 1922 con *E. Norvegicus,* haciendo las pruebas por inoculación cutánea y subcutánea.

Posteriormente, los trabajos de Malone, Avaria y Naidu demuestran una clara relación entre la susceptibilidad o la inmunidad de la rata a la peste y el poder bactericida de su sangre, y consideran, además, que la fagocitosis interviene esencialmente en el proceso.

Es más: los estudios bacteriológicos y anatomopatológicos que llevó a cabo la "Comisión Inglesa" en las ratas de las zonas pestosas están también en relación con estos hechos globales que acabamos de describir relativos a la existencia o inmunidad de las ratas.

En efecto, la Comisión acepta, desde luego, la existencia de lesiones de peste crónica, pero concluye que tiene poca o ninguna significación epidemiológica, y los estudios histológicos de Ledingham les condujeron a la conclusión de que las lesiones se encuentran realmente *en un proceso de regresión y de curación.* Por esto creó entonces el término de *resolving* o *de resolved plague,* ya que este término evitaba el concepto erróneo de "peste crónica" y expresaba más exactamente el curso del proceso.

Este cuidadoso estudio demostró que no hay una franca línea de separación entre peste aguda, subaguda, *resolving* y *resolved,* y que puede ser trazada una serie de continuos cambios patológicos hacia el período o *estadio final de curación.*

Ledingham concluyó que la reacción de las células del tejido fijo del bazo pueden llegar a la completa encapsulación de los abscesos producidos por la invasión del órgano por el bacilo.

Estas lesiones y estadios de peste *resolving* fueron estudiados primero en las ratas infectadas espontáneamente, y después, experimentalmente.

Finalmente, la Comisión no pudo observar un solo caso de transformación de peste *resolving* o *resolved* en peste aguda, considerando, además, que un tal caso es improbable que pueda ocurrir por el hecho de que durante el proceso de *recovery* la rata adquiere una inmunidad considerable.

Se ha sugerido además que el bacilo puede ser atacado por los fagocitos de la misma sangre, ingerida en el proventrículo de la pulga, y que, a temperatura alta, la fagocitosis es más activa. Esto podría explicar el por qué una pulga infectada se desembaraza más rápidamente de los bacilos a temperatura alta que a temperatura baja, y si es alimentada con sangre normal que si se mantiene en ayunas después de infectada. Pero lo que es más importante en relación con lo que llevamos dicho anteriormente es que la pulga se libra aún más rápidamente de los gérmenes cuando se alimenta sobre animales inmunizados. Por lo tanto, la creciente proporción de ratas inmunes que tiene lugar al finalizar una epizootia puede ejercer un efecto inhibitorio en la expansión de la enfermedad.

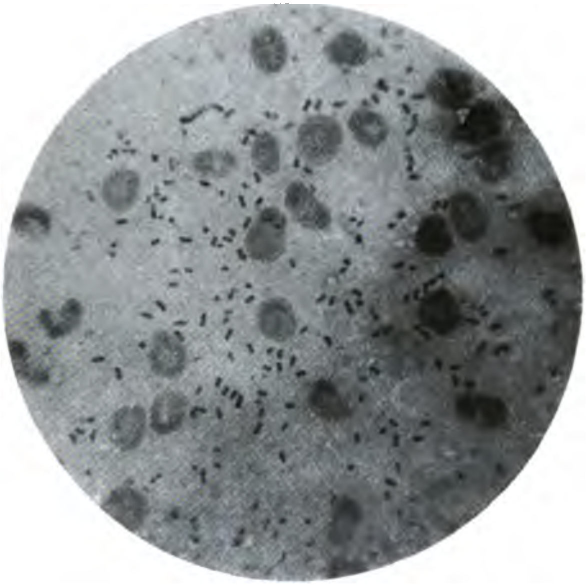
Un nuevo factor puede contribuir al mismo fin, y es la disminución de la virulencia del *bacilo pestis* en el organismo de las ratas inmunizadas. En efecto, Burgers (1930) cita sus experiencias, durante las cuales la virulencia del B. pestoso ha sido abolida o atenuada por pases en ratas inmunes o parcialmente inmunes. Una raza de *B. pestis,* atenuada por el pase a través de un animal inmunizado, reprende su virulencia por pases por animales normales, pero mucho más lentamente que un cultivo atenuado por envejecimiento, ya que se necesitan cinco pases sobre animal sano en el primer caso, y mientras que es suficiente uno solo en el segundo. (Burgers: *The Journal of Higiene,* 1930.)

Podríamos añadir aún que puede concurrir al mismo resultado la intervención de un bacteriófago antipestoso en las ratas infectadas. D'Herelle, en 1921 y en 1925, aisló un bacteriófago activo de las heces de ratas capturadas en cuatro distintos sitios de una ciudad de Cochinchina, en la que hubo un pequeño brote de peste humana; estas ratas habían resistido probablemente a la infección, y el bacteriófago se encontró en cada uno de los cuatro grupos de ratas.

Después de cien pases, 0,02 de cc. del bacteriófago, lisaban completamente 10 cc. de un cultivo en un caldo de *B. pestis* de veinticuatro horas.

Estos hechos de la resistencia que adquieren las ratas a la infección pestosa (inmunidad) durante el curso de una epizootia, inmunidad que se transmite a las nuevas generaciones y que motivan la sustitución de la peste aguda, septicémica (única transmisible a los ectoparásitos), por las lesiones con tendencia a la curación, que constituyen la "resolving" y "resolved plague", con la cooperación de la eliminación de los ejempla-

LAMINA 11



Bacilo pestoso aislado de uno de los enfermos de la epidemia de 1931 en Barcelona.
(Microfotografía *Domingo-Cartañá.)*

res e individuos más sensibles en el período agudo de la epizootia (lo que nos permitiríamos llamar "auto-desratización", la posible intervención del bacteriófago, la fagocitosis ejercida dentro del proventrículo de la pulga y, además, la desratización activa practicada por el hombre por todos los medios, pueden explicar la desaparición de una epizootia en focos reducidos y limitados, tales como, por ejemplo, los colindantes de las modernas ciudades, y, en el caso presente, los de Barcelona.

Epidemia de 1931. Consideraciones sobre el desconocimiento de su origen.

Los hechos citados anteriormente nos inclinan a pensar que el virus de este brote epidémico fué importado; sin embargo, cabe la sospecha, y es admisible, de que tuviera relación de dependencia con los casos que se observaron el año anterior, por la razón de que el período que medió entre los dos no es excesivamente largo (octubre de 1930 a agosto de 1931); y aun podría admitirse además, en este caso, una relación de contigüidad, puesto que el pequeño foco de 1930 puede situarse en la Carretera de la Bordeta, 56, y el principal foco de 1931 fué en Hospitalet (sitios relativamente próximos: véase el mapa).

Admitiendo esta relación, no por eso tenemos que dejar la idea de la importación del virus, que tendríamos que situar entonces en 1930; mas como no ha sido posible encontrar ni la puerta de entrada ni mucho menos la fecha en que pudo ocurrir, todas las conclusiones que debamos sacar han de ser hipotéticas, y deben ser razonadas por deducciones epidemiológicas.

Son cuatro los principales factores que deben analizarse como posibles causantes de la entrada del virus, aunque con muy diferente probabilidad en cada uno de ellos:

a) Animales infectados (roedores u otros).
b) Materiales contaminados.
c) Pulgas infectadas (sin roedores).
d) Hombre como portador de virus.

a) Animales infectados.

La posibilidad de roedores infectados como importadores del virus en un lugar determinado está fuera de duda, y tan clásico es el conocimiento del transporte del virus por las ratas pestosas como principal vehículo activo, que les ha merecido el calificativo de "Viajantes de la peste".

Otra modalidad la constituye el ingreso del virus con estos animales muertos; a este efecto, convendrá recordar que, si bien es cierto que el bacilo pestoso, por lo general, es lábil en la competencia con los otros gérmenes que pululan en el cadáver (gérmenes de la putrefacción), hasta el punto de que en el laboratorio el aislamiento del germen en un animal inoculado experimentalmente conviene hacerlo pronto, so pena de correr el riesgo de ver el resultado negativo), no lo es menos que en ciertas condiciones su vitalidad en el cadáver pestoso puede ser larga.

De los trabajos de Kister y Schumacher (1905) se desprende que la posibilidad de encontrar el bacilo pestoso en los cadáveres de las ratas depende de tres factores principales, que son: la temperatura ambiente, la cantidad de bacilos y el grado de putrefacción.

Estos autores pudieron encontrar el bacilo en un cadáver de noventa y tres días sometido a una temperatura de 5 a 12 grados, pero solamente a los cuatro días si la temperatura había sido de 32 grados.

Zlatogoroff ha podido encontrarlo hasta los *ciento nueve días* en el cadáver a temperatura de tres a cinco grados.

b) *Materiales contaminados.*

La vitalidad del bacilo pestoso es débil en los cultivos (poca resistencia a la temperatura, a la luz, desecación, antisépticos, etc.).

En los productos patológicos su resistencia es ya mayor (pus, esputos, etc.), sobre todo preservados de la luz y con humedad conveniente; en estas condiciones ha podido encontrársele vivo, ya en el suelo o en vestidos contaminados al cabo de algunos meses.

Como ejemplos de vitalidad larga, citaremos los siguientes:

Francis publica en 1933 sus resultados sobre el particular, diciendo que un fragmento de bazo de cobaya pestoso, conservado en glicerina pura a 15 grados, ha conservado su virulencia durante siete años, y que un tubo de agar sembrado con *B. pestis* y cerrado a la lámpara y guardado a 10 grados ha conservado su virulencia durante nueve años.

En sus publicaciones de 1928, Nikanoroff dice que ha comprobado que el "kochma" (especie de manta que usan los indígenas de las estepas del SE. de las Repúblicas Soviéticas), manchando con esputos bacilíferos (peste neumónica), puede conservar los bacilos vivos durante cinco meses y medio, cuando este tejido es mantenido en la obscuridad y preservado de la desecación durante los meses de verano al ser enrollados y guardados para el invierno siguiente.

c) *Pulgas infectadas.*

La posibilidad de que sean las pulgas solas las que vehiculen el bacilo pestoso, transportándolo a puntos muy distantes, está fuera de duda. A este efecto, citaremos los siguientes datos.

Longevidad de las pulgas sin infectar.

Los estudios experimentales de Bacot, alimentando artificialmente a pulgas sobre el hombre o animales, dan las cifras siguientes, variables según las diferentes especies.

	Alimentadas	No alimentadas
Pulex irritans....................	513 días.	125 días.
Ceratophyllus fasciatus.......	106 —	95 —
Xenopsylla cheopis............	100 —	38 —
Ctenocephalides canis.........	234 —	58 —
Ceratophyllus gallinae.........	354 —	127 —

Y aun el mismo autor concluye que, cuando la alimentación no puede ser regular, sino muy espaciada por la falta de huésped, la sobrevivencia puede ser la siguiente:

Ceratophyllus fasciatus......................	22 meses.
Pulex irritans..................................	19 —
Xenopsylla cheopis...........................	10 —
Ctenocephalides canis........................	18 —

Infectividad: Se ha estudiado por diferentes autores el límite de la duración de la infectividad en las pulgas.

Otten, en Java, halló experimentalmente que era de cuarenta y tres días para *Xenopsylla cheopis.*

Hoeseu, en Java también, y para la misma especie, no consiguió pasar de los treinta y tres días.

Svellengrebel fijó el límite máximo de la infectividad para la *Xenopsylla* en sus experiencias en veintiocho días, a 23 grados y 76 de humedad.

Raybaud y Gauthier (1911) llegaron a los cuarenta y cinco días con la *Ceratophyllus* a la temperatura de la helera; y

Bacot, practicando las mismas experiencias a temperaturas variables, cuya media era de 7,5 grados, obtuvo el límite de la infectividad a los veintinueve, treinta y cuatro y cuarenta y siete días respectivamente.

Pero los recientes datos del profesor Nikanoroff, director del Instituto de Microbiología y Epidemiología del S. E. de la U. R. S. S. (1928), dan cifras verdaderamente insospechadas que sobrepasan, creemos, todos los resultados obtenidos y conocidos. Este autor afirma que operando con una especie de las pulgas del espermófilo (*Ceratophyllus tesquorum, Neopsylla setosa* o *Ctenophthalmus breviatus*), se comprobó la conservación del B. pestoso en la pulga viva durante *doscientos seis días,* y es probable, dice, que no sea aún el término de su vitalidad. Además, la virulencia del B. pestoso al cabo de una tan larga permanencia en el organismo de la pulga no disminuye. En consecuencia, dice el autor, la pulga conserva el germen de una estación a otra (período de invernación), al igual que el espermófilo.

Según sus experiencias, la pulga puede vivir diez meses sin alimento. Estos sorprendentes resultados del Prof. Nikanoroff sobrepasan todos los alcanzados que nosotros hemos obtenido. Nuestros resultados experimentales en cuanto a longevidad con pulgas alimentadas y no alimentadas son de todo punto inferiores, y aun debemos confesar que no han sido fáciles de obtener, pues los fracasos (muerte de los ejemplares a los primeros días de experiencia) era lo más frecuente, obligándonos docenas de veces a una paciente y enojosa repetición. (Véase la técnica seguida por nosotros en otro capítulo). Conviene tener en cuenta que las observaciones del Prof. Nikanoroff fueron practicadas en lugares donde las temperaturas son extremadamente bajas y que, por lo tanto, permitirían una longevidad mayor en las pulgas no alimentadas y un período de infectividad mucho más largo también que el que se observa en los países de temperaturas altas o moderadas.

d) Hombre como portador.

En estos últimos años se han descrito numerosos casos de portadores: Leger y Baury, 1923; Tanon y Camtessedes, 1923; Uriarte, 1924; Durand y Conseil, 1927; Boston, 1927. Duran y Conseil y otros autores sugieren la posibilidad de que los portadores pueden durante largo tiempo guardar el bacilo sin manifestaciones clínicas, pero que la ocurrencia de otra enfermedad infecciosa (gripe o sarampión, por ejemplo), provocando un descenso en sus defensas hace posible la aparición de una septicemia pestosa, con la posibilidad de la expansión de la infección por pulga, etc., o por vía respiratoria; en otras palabras, constituir el punto de partida de una epidemia.

Recientemente Nikanoroff confirma este hecho diciendo que ha sido confirmada la septicemia por *B. pestis* en personas aparentemente sanas o en convalecencia y que, naturalmente, ellos constituyen la fuente de infección por medio de pulgas.

A Petrie le parece todo esto de una exagerada liberal interpretación, diciendo que hacen falta más observaciones bacteriológicas y epidémicas para aceptarlo.

Nikanoroff cita un caso que observó en 1923 de un kirgis portador de una adenitis pestosa y que en seis semanas, habiendo convivido con tres familias diferentes, en cada una de ellas se produjo un caso de peste al cabo de pocos días de su llegada.

Durand y Conseil, a quienes hemos mencionado, citan un caso de un portador de *B. pestis* durante diecisiete meses. La existencia de tales portadores explicaría (por el mecanismo que indicamos antes de la concurrencia de una enfermedad anergizante) la reviviscencia de las epidemias al cabo de varios meses, y tal importancia conceden dichos autores a este hecho, que comenzaron el estudio de la intradermorreacción con el bacilo pestoso calentando a 100°, con objeto de despistar los posibles portadores.

La noción clásica de la rata como "*único reservorio del virus*" pierde valor y debe tenerse en cuenta también al hombre sano como un posible reservorio, *en tanto que portador*, concluyen estos autores.

No puede negarse la posibilidad de la intervención de alguno o algunos de estos factores citados. Pero después del estudio de todos los datos que pudieran relacionarse con cada uno de estos cuatro factores y de que dispusieron las autoridades sanitarias, y no pudiendo no sólo hallar, sino tan sólo entrever la posibilidad del hallazgo de la puerta de entrada, y dada además cierta relación, aunque no muy clara, de algunos casos con comercio de trapos, podría sacarse la conclusión de que lo más probable es que el virus entrase con pulgas infectadas, que, introducidas en el interior de balas, embalajes o cargamentos de mercancías, pueden escapar perfectamente a las más estrechas medidas de vigilancia sanitaria o desinfección, etc. Pero repetimos que esta deducción es hipotética y que la puerta de entrada de la peste de 1930-31 ha sido desconocida.

Epoca del año en que se han presentado brotes epidémicos de peste bubónica en Barcelona.

1905-1906.—De junio de 1905 a abril de 1906.

Máximo número de casos, en julio de 1905 y noviembre y enero de 1906.

1919.—Septiembre, y un caso en los primeros días de octubre.
1922.—Septiembre, octubre y noviembre.
1923.—Octubre y noviembre.
1930.—Octubre.
1931.—Agosto, septiembre, octubre, noviembre.

Relación de estas epidemias con nuestros estudios sobre las pulgas.

Vemos que, exceptuando los casos que se observaron en invierno de 1906, todas las epidemias habidas en Barcelona han tenido lugar durante los meses de julio a noviembre. (Descartamos el caso del año 1920 y el del 1925 por ser de origen exógeno.)

Si lo comparamos con los resultados de nuestros estudios sobre los ectoparásitos de las ratas de Barcelona, veremos que coinciden con el período de máximo número de *Xenopsylla cheopis*.

Aunque el solo hecho de orden entomológico a que aludimos (véase gráfico de la fig. 4) es suficiente, dados los conocimientos que se tienen de la transmisión de la peste por la *Xenopsylla,* la coincidencia con estos otros datos confirma y explica el porqué de las epidemias habidas, llevando a la conclusión, ya bien conocida por la experiencia, *de que el período del año de peligro para la peste en Barcelona es el verano, y el otoño sobre todo.*

Los casos de enero de 1906 pueden explicarse por condiciones excepcionales de temperatura alta (que hubiesen permitido un incremento de la *X. cheopis)* o que se verificara la transmisión por *Ceratophyllus fasciatus* (la pulga más numerosa en invierno en Barcelona) y que puede también transmitir la enfermedad.

Campaña sanitaria durante la epidemia de 1931.

El servicio sanitario actuó durante este tiempo en la siguiente forma:

a) Denuncia del caso sospechoso al Instituto Municipal de Higiene.

b) Comprobación clínica por un médico de dicho Instituto.

c) Traslado del enfermo y convivientes al Hospital de Infecciosos.

d) Diagnóstico bacteriológico.

e) Aislamiento del enfermo en un cuarto individual. Los convivientes quedaban en observación durante siete días en un pabellón del mismo hospital, habilitado para tal fin. Durante este tiempo, eran vacunados y se practicaba una activa desratización y desinfección de sus casas, que quedaban selladas hasta que volvían sus ocupantes. Para facilitar la denuncia y evitar que pudieran quedar algunos casos sin diagnóstico, se cursó una circular a todos los médicos, recordándoles el cuadro sintomático de la peste y la necesidad de la declaración urgente.

La comprobación clínica y el traslado del enfermo y de los familiares convivientes quedaban efectuados inmediatamente, circulándose las órdenes por teléfono. A su llegada al Hospital de Infecciosos eran rapados y bañados en el pabellón respectivo.

El examen bacteriológico consistía en la punción del ganglio y siembra de dos tubos de agar y un tubo de caldo, haciendo a la vez un par de frotis. A partir de los cultivos se hacía la inoculación intraperitoneal al cobaya.

Los enfermos fueron tratados específicamente con suero del Instituto Nacional de Higiene, del Instituto Pasteur de París y del Laboratorio Municipal de Barcelona, con cantidades que oscilaban entre 60 y 120 cc. diarios. La campaña sanitaria que se desarrolló tuvo por principales objetivos el despistir los casos nuevos prontamente; la vacunación del mayor número posible de los habitantes de las zonas infectadas; la desratización activa de dichas zonas; la desinsectación de todos los locales públicos y casas particulares; el estudio de la solución del tratamiento de las basuras domiciliarias, que es, a nuestro modo de ver, la causa principal y responsable del problema sanitario que estudiamos. Para la vacunación dividimos Hospitalet en tres sectores con un Dispensario en cada uno de ellos y una Oficina Central, y funcionaba además una sección móvil de vacunación para ir a los barrios alejados, masías, etc. Vacunáronse en total unos 4.000 individuos. La desratización y desinsectación se realizaron como a continuación exponemos:

Desratización.

Brigada eventual.—Durante el brote epidémico de peste de 1931, la necesidad de intensificar la desratización hizo aumentar el personal de los equipos de desinfección del Instituto Municipal de Higiene para verificar la desratización en Barcelona. Asimismo, el Municipio de Hospitalet creó una pequeña brigada, que actuó dirigida por un empleado del Instituto Municipal de Higiene y con material de éste.

En 30 de noviembre de 1931 empezó la desratización de Hospitalet con fondos suministrados por el Estado (5.500 pesetas) y la Generalidad (12.000 pesetas), con un equipo a las órdenes de un capataz del Instituto Municipal de Higiene, y se instaló un centro para depósito de materiales, confeccionar las comidas envenenadas, etc. Con este dinero se llevó la campaña de desratización (que ahora detallaremos) desde 30 de noviembre de 1931 al 30 de junio de 1932. El día 25 de junio el Estado concedió una nueva subvención de 10.000 pesetas, y la Generalidad de 12.000.

Brigadas.—Actualmente el Ayuntamiento de Barcelona tiene una brigada encargada de la desratización, integrada por 58 empleados repartidos en cuatro Centros:

Centro de San Martín: Antiguo Matadero de San Martín. Calle de Espronceda.

Centro Sur: En el Palacio de Agricultura de la Exposición.

Centro Oeste: Sarriá. Calle del Capital Mirambell.

Centro Norte: Gracia.

Raticidas empleados. Virus de Danzy (facilitado por el Laboratorio Municipal en los sitios donde se críen animales domésticos):

Pasta fosfórica Steiner (fósforo disuelto en una grasa que se tiñe con azul de metileno).

El alimento con que se han empleado estos raticidas ha sido el pan, del día anterior (para mayor consistencia) cortado en pequeños cubitos (de unos tres centímetros de lado).

Se colocan sobre pedazos de papel blanco para poder contar los trozos colocados y recontarlos a la mañana siguiente, y deducir los que han sido devorados.

El pan con fósforo se repartía por las tardes, y el virus de Danyz, al anochecer. Al propio tiempo se han colocado rateras para capturar ratas vivas para el estudio de los ectoparásitos y las investigaciones de laboratorio necesarias.

Los datos detallados de esta campaña de desratización han sido publicados por el director de los servicios municipales de desinfección de Barcelona, Dr. Claramunt, actual director del Instituto Municipal de Higiene (1), por lo que no daremos más que un extracto del trabajo diario de estas brigadas, resumimos de dicha Memoria.

Extensión de la zona desratizada en Hospitalet: 1.314 hectáreas. En ella están enclavados los principales focos de población murina por razón del tráfico de basuras que allí existe.

Según los datos exactos que nos da el Dr. Claramunt, se han desratizado 6.223 locales (casas, fábricas, almacenes) con pasta fosfórica; 1.138 con virus Danyz; 170 masías, 70 establecimientos industriales, 57 barracas, 181 almacenes de basuras y 7 almacenes de trapos.

Se trataron tres veces el cementerio de Hospitalet; tres veces el de Sans; 13 veces la Remonta de Caballería, y cuatro los almacenes de la Campsa.

Se siguieron tres veces las márgenes de los canales, acequias, torrentes, cañaverales, terraplenes y los márgenes entre los campos de cultivo.

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(1) Véase *Revista dels Serveis Sanitaris i demografics Municipals de B.* Año IV, número 11 (1932).

CAPITULO X

ESTUDIO DE LAS ZONAS DE DEPOSITO DE LAS BASURAS DE BARCELONA

Por tener la convicción de que el brote epidémico mencionado era de origen murino y por ser los focos de ratas consecuencia inmediata del actual sistema de aprovechamiento de basura, nos decidimos a hacer un pequeño estudio sobre las de Barcelona durante nuestra estancia en Hospitalet.

La afirmación de que el brote citado fuera de origen murino no descarta la intervención de los trapos. En las mismas zonas de los depósitos de basura, y por razones de conveniencia comercial, existen la mayor parte de los traperos mayoristas. Nadie podría negar, pues, la posibilidad de que la peste fuera importada por pulgas o ratas que vinieran con los trapos de zonas infectadas; pero, aparte de que esto no ha podido comprobarse, hay que contar con la epizootia murina, pues de lo contrario, tendríamos que admitir que el contagio de la peste bubónica que relatamos fué interhumano, cosa insospechable, porque no se ha visto la relación y dependencia de unos casos con otros mientras que están bien establecidas las relaciones de la mayor parte de los enfermos con los focos de basuras y, por ende, su convivencia con las ratas.

En la exposición de esta encuesta nos referiremos solamente a las basuras domiciliarias, ya que las restantes, por su escasa importancia, quedan incluídas en el servicio de limpieza de calles.

En Barcelona el servicio de recogida y aprovechamiento es el siguiente: El Ayuntamiento tiene un contrato directo con la Sociedad "Fomento de Obras y Construcciones", entidad que a su vez tiene unos subcontratos con cada uno de los societarios de la "Cooperativa de Usuarios del servicio de limpieza domiciliaria de Barcelona", que son los que realizan este trabajo. De manera que "Fomento de Obras y Construcciones" es, en realidad, una entidad contratista intermediaria entre la "Cooperativa" y el Ayuntamiento. Esta "Cooperativa" está integrada por los profesionales basureros ("escombriaires"), que forman un grupo de familias que ya desde unos ciento cincuenta años vienen prestando este servicio a la ciudad. La finalidad de esta asociación es la de obtener un mayor rendimiento con la venta en común del producto de selección de las basuras (trapos, papel, lata, etc.), que haciendo la venta parcial e individualmente Los socios que constituyen esa "Cooperativa" son 166, repartidos entre los distintos barrios siguientes: Coll Blanch, 15; Casa Antúnez, 14; San Martín, 30; Casa Pi, 50; Canyet y Santa Eulalia, 57.

Cada usuario dispone de un carro y percibe 14 pesetas diarias. Hacen dos carretadas diarias, o sea 332 carretadas, que, a razón de tres metros cúbicos de cabida, son 996 metros cúbicos, y como no sería suficiente este servicio, funcionan además diariamente 100 carros auxiliares o más, que hacen un total de 561 carretadas diarias, o sea 1.683 metros cúbicos de basuras.

Cada *escombriaire* tiene su demarcación señalada, de la que recoge su basura diariamente. Las basuras son trasladadas con los carros reglamentarios a los sitios de depósito, que están situados en los diferentes barrios que hemos indicado anteriormente, y que están en su mayoría en la parte sur de Barcelona (Hospitalet y Casa Antúnez) y otro pequeño grupo en San Martín (parte norte). Dichos carros, a la salida de Barcelona, tienen un control de la guardia urbana, para evitar que contravengan lo estipulado en el contrato, tal como número de viajes, exceso de carga, prohibición de llevar nada fuera del carro, que debe ir herméticamente cerrado, etc.

Los sitios de depósito de las basuras en Hospitalet están generalmente constituídos por un amplio solar cercado de tapia, en uno de cuyos lados hay la habitación del basurero, la cuadra para la caballería y el corral de los cerdos. La mayor parte, pues, de este cercado está constituído por el solar donde se descargan y se acumulan a cielo abierto las basuras que diariamente se aportan.

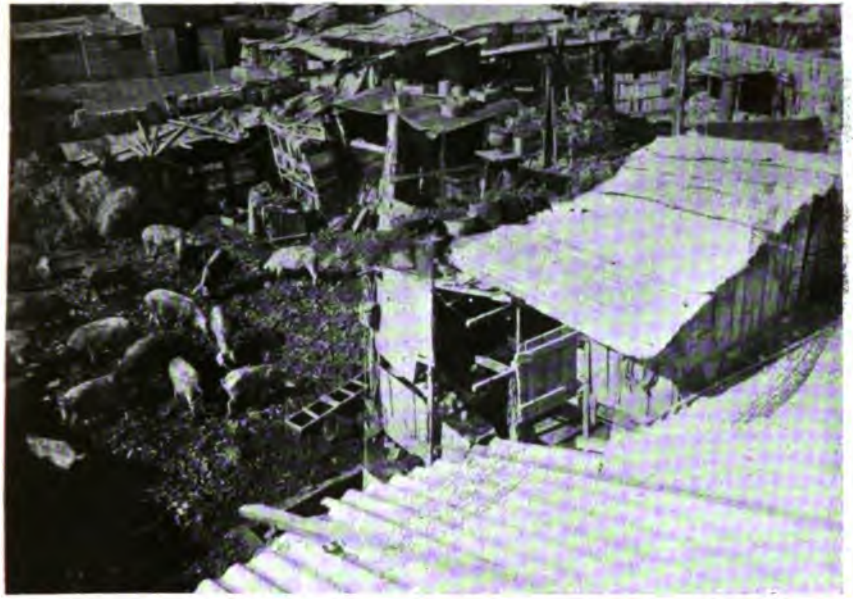
Existen además otros basureros, a los que se les llama "campbuscaires", que no tienen contrato alguno ni constituyen agrupación organizada y carecen de carros reglamentarios. No tienen, por tanto, tampoco demarcación señalada en la ciudad, y recogen las basuras de donde pueden para poder luego con ellas dedicarse a la cría de puercos y a la selección y venta de los productos obtenidos. El número de estos "campbuscaires" es de 132, distribuídos del modo siguiente: Coll Blanch, 25; Casa Pi, 60; Canyet, 22; San Martín, 20; Casa Antúnez, 5.

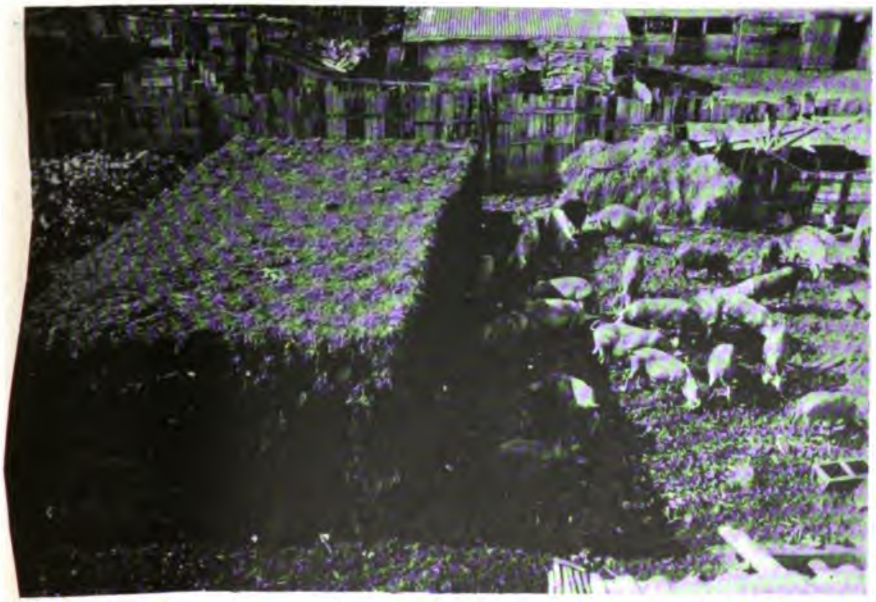
Destino y utilización de estas basuras.

Se sigue el sistema de aprovechamiento total de la manera siguiente:

a) Cría de cerdos con los restos alimenticios.

b) Selección y reventa de todos los desperdicios aprovechables.





Dos aspectos de los depósitos de basuras de Hospitalet, en donde se dieron casos de peste en 1931. Se ven las habitaciones humanas contiguas.

c) Utilización como abono orgánico (estiércol) de todo el material restante no aprovechable.

El funcionamiento es el siguiente: El carro vacía sus dos cargas diarias en medio del cercado, sueltan entonces los cerdos, que consumen con todo lo apetecible el primer turno de la selección; son luego encerrados de nuevo en las porqueras, y entonces se practica la selección cuidadosa de todo lo aprovechable (vidrios, zapatos, alpargatas, trapos, papel, latas, cueros, maderas, pan, huesos, metales, etc.), que se van separando en pequeños montones para ser vendidos luego a las industrias correspondientes (fundiciones de hierro, papeleras, traperos al por mayor, fundiciones de vidrio, etcétera). A continuación indicamos algunos de los principales industriales o comerciantes a los que van destinados los trapos, papel, huesos, vidrios, alpargatas, etc.

Trapos.

Pantinat, Aprestadora y Angel Guimerá.
Valentín Arolas, Igualdad.
Remigio Mira, Angel Guimerá.
Enrique Armengol, Aprestadora, detrás Paulino.
Ramón Castelló, Pasaje Elías.

Papel.

Papelera del Prat, Sociedad Anónima, Carretera del Prat.
Viuda de Quirico Casanovas, Casa Antúnez.
N. Renóm, Calle de Sarriá (Barcelona).
Planas, Aragón (junto a Dos de Mayo).
Huesos.—Valentín Arolas, Calle de la Igualdad (Canyet).
Vidrios.—Remigio Mira, Angel Guimerá.
Alpargatas.—Coll de Mont, Maluquer (Santa Eulalia).

Para el aprovechamiento de la lata, la Cooperativa dispone de unos hornos, donde se la hace perder el estaño, y luego se prensan para venderlas a las fundiciones de hierro. Con esto se evita la cantidad enorme de latas que sembraban antes los campos vecinos de las zonas de los basureros. El rendimiento de esta selección es de unas 105 pesetas semanales para cada basurero, sin contar con el beneficio que les proporciona la cría de los cerdos. El número total de cerdos que se crían con los restos alimenticios de las basuras asciende entre todas las zonas a 10.600. Así, pues, como vemos, casi todo lo que sale de la ciudad constituyendo las basuras vuelve a ella debidamente clasificado, para ser aprovechado por las industrias respectivas, o ya completamente transformado, como en el caso de los desperdicios alimenticios convertidos unos meses después en carne de cerdo. (Véase figura 7.) El hecho tiene su importancia desde el punto de vista epidemiológico, mostrando las relaciones que existen entre estas zonas de basureros y la ciudad y el peligro de que en tiempo de epidemia puedan aquellos productos vehicular la infección a puntos distantes y ser responsables de casos aparentemente desligados de la zona infectada. El último residuo lo constituye, como hemos dicho, el estiércol, que es vendido principalmente a los agricultaros de la Cuenca del Llobregat. Todos los productos sépticos de hospitales, sanatorios, dispensarios, etc., son recogidos aparte y quemados en unos hornos especiales. Además, se echa directamente al mar una cierta cantidad de basura con dos gánguiles y un remolcador, que a este efecto tiene la Sociedad contratista; los carros que van a dichos gánguiles vierten directamente en ellos su carga, sin que se constituya un montón de basura de descarga en el muelle. Estas basuras se vierten a seis kilómetros de la costa.

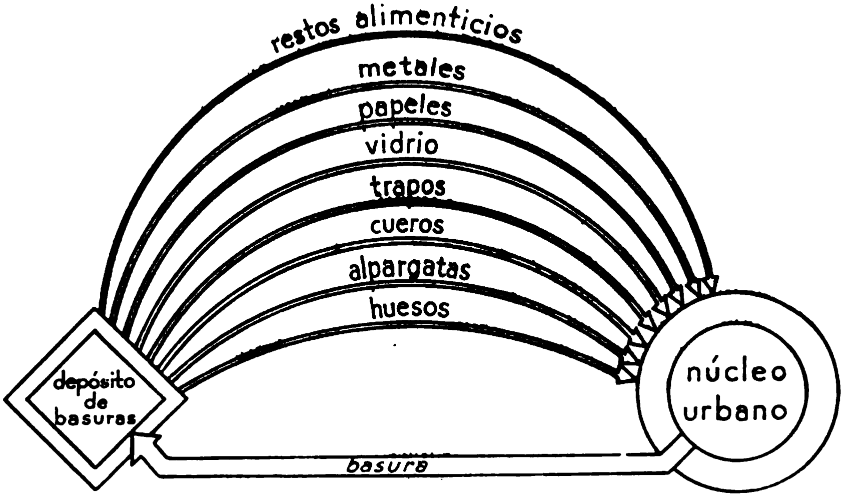


Fig. 7.—Esquema de la clasificación de la basura domiciliaria para su devolución al núcleo urbano.

Esta breve exposición y la observación de las fotografías que acompañamos indican claramente que *estas zonas reúnen todas las condiciones para la vida de los roedores, que tienen alimento en cantidad y calidad más que suficiente y abrigo a propósito.*

La convivencia entre todos los seres que viven a expensas de la basura es absoluta (hombre, cerdo, rata, perros, gallinas, pulgas, mosca, etc.). En algunos sitios (los peores) la habitación humana está construída, como se ve en las fotografías, con trozos de chapa, restos de madera, etc., a planta baja y casi sin separación ninguna con el centro del solar constituído por un montón de estiércol y basura de todas clases. Todas las circunstancias que intervienen en la epidemiología de la peste concurren pues, aquí. En verano, cuando las condiciones de temperatura y humedad son favorables, es un criadero de pulgas, de tal manera, que los enfermos que nosotros hemos tenido ocasión de ver están completamente tatuados de picaduras de estos insectos.

No es de extrañar, pues, que sean estas zonas las más peligrosas en cuanto a peste, y que sea en ellas donde se desarrollaron el año 1931 la casi totalidad de los casos.

Estas pésimas condiciones no se dan en todos los depósitos de basuras, sino casi únicamente en los "campbuscaires", que son lo que viven peor, la mayoría forasteros, gente menesterosa, sin profesión determinada, que acuden a la capital huyendo del hambre de sus pueblos. Los basureros asociados que constituyen la Cooperativa viven mejor instalados, habitando casas bien acondicionadas, piso alto completamente separado del montón de basura y de los establos, porquerizas y cuadra para los caballos. Algunos de ellos están, desde el punto de vista de granja agrícola, en buenas condiciones; por ejemplo: el grupo de casas que constituyen el pasaje Carbonell, con sus porquerizas bien instaladas, bar, escuela, etc., adoquinado de la calle central, cloacas, pozo con bomba para cada basurero, etc. Asimismo algunas de las casas de los otros "usuarios" del grupo de Santa Eularia, etc. A pesar de esto, dejan todas que desear, pues están bien construídas con fines de aprovechamiento agrícola, pero mal como *instalaciones de orden sanitario.*

Los almacenes de trapos abundan también en pulgas, y no tanto en ratas. Aunque en todos ellos se encuentran estos roedores, porque están en su mayor parte situados en pleno dominio de la población murina (barrios donde están enclavados los basureros).

Posibles soluciones al problema de las basuras.

Lo expuesto es más que suficiente para demostrar que el actual estado de cosas requiere urgentemente una solución de acuerdo con los principios sanitarios para evitar el peligro que actualmente supone.

Comenzaremos este capítulo con el adjunto esquema, tomado del libro del ingeniero sanitario Sr. Paz Maroto.

Sistemas de tratamiento de las basuras......
  1) Aprovechamiento.
  2) Destrucción.

1.º Aprovechamiento.
  a) Total... (El actualmente seguido en Barcelona: cría de cerlos, selección, estiércol).
  b) Parcial.
    Trituración o Fermentación.
      Para destinar el producto resultante a la agricultura (abono orgánico).

- 2.° Destrucción.
  - a) Vertido simple......
    - Al mar.
    - A marismas o terrenos pantanosos.
    - A barrancos u hondonadas.
    - A socavones mineros.
    - A terrenos inundables por la pleamar, etcétera.
  - b) Cremación total......

Desechamos, por los inconvenientes expuestos, el sistema actual de aprovechamiento total y el de destrucción por vertido simple. No es necesario esforzarnos para demostrar que el vertido al mar no podría ser una solución para Barcelona, pues, aparte del coste elevado, bastará indicar que solamente dos días de temporal que impidiese la salida de los gánguiles implicarían un almacenamiento de 3.200 metros cúbicos de basuras en el muelle de Barcelona, de manera que con este método quizás no se conseguiría otra cosa que trasladar el centro de basuras de su emplazamiento actual al puerto de nuestra ciudad, modelo de orden sanitario.

Nos quedarían, pues, para elegir, los dos sistemas de aprovechamiento parcial (trituración y fermentación) y el de destrucción total (incineración).

El de trituración consiste en la reducción de las basuras a polvo mediante unos aparatos llamados masticadores, que, gracias a la acción combinada de desintegración y mezcla con un funcionamiento a gran velocidad, reducen las basuras a un polvo inerte y sin olor. Cada masticador puede triturar seis toneladas de *basura seca* por hora, y necesita un motor de 50 caballos. Hay casas constructoras de masticadores para materias húmedas, tales como desperdicios de matadero, etc. Las ventajas de este tratamiento consisten, sobre todo, en la baratura de instalación y entretenimiento.

El procedimiento de fermentación se basa en la utilización como abono orgánico del producto resultante de la descomposición de la materia orgánica en cámaras zimotérmicas.

La fermentación en estas cámaras (vaso cerrado) produce temperaturas que llegan a 70 y 80° durante quince y veinte días o más que dura el tratamiento, suficientes, pues, para la destrucción de los gérmenes patógenos, insectos, larvas y huevos, semillas de plantas parásitas, etc. El producto resultante es mucho más rico (el doble aproximadamente) en elementos fertilizantes que el estiércol, cuya fermentación se ha hecho al aire libre con grandes pérdidas. Constituye una masa porosa de una gran utilidad para la agricultura, no sólo por el abono químico que representa, sino por sus cualidades físicas, que hacen que retenga el agua, actuando de esponja y facilitando la duración del contacto de todos los principios solubles nutritivos para los vegetales. Así, pues, la materia resultante de esta transformación biológica es elemento inmejorable para lo que en agricultura se llama corrección de las tierras.

El sistema de cremación destruye por este procedimiento las basuras en hornos especiales y utiliza el calor así producido para transformarle en electricidad, que es vendida luego a la industria. Asimismo, aprovecha las escorias resultantes (para fabricación de ladrillos, por ejemplo), que representan un 25 por 100 del tonelaje total.

Aunque la basura, en general, es un combustible pobre, la recuperación de este poder calorífico por su transformación en electricidad puede dar un rendimiento utilizable.

Por término medio, se da como rendimiento unas 1.000 calorías en verano y 3.000 en invierno por cada kilogramo de basura. Según Paz Maroto en nuestras poblaciones la incineración de un kilogramo de basura puede producir de 0,8 a 1,20 kilogramos de vapor, y en cuanto a energía eléctrica, una tonelada de basura puede producir 120 a 180 kilovatios-hora. Según este cálculo, la incineración de las basuras de Barcelona podría dar el siguiente rendimiento. Pongamos, por ejemplo, que Barcelona tenga que quemar 500 toneladas diarias (cifra, como hemos visto, inferior a la realidad); 500 toneladas a 120 kilovatios serían 60.000 kilovatios diarios, que al año darían 12.900.000 kilovatios, que, pagados a 0,10 pesetas, serían 1.290.000 pesetas.

Esta cifra, a primera vista, puede dar una impresión optimista, pero es necesario tener también en cuenta el coste de la fabricación de esta electricidad. Para evitar entrar en detalles sobre este punto, creemos lo mejor resumirlo todo, dando a continuación una nota de los gastos de instalación y entretenimiento que implican la fermentación y la incineración, que hemos leído en el dictamen de la Comisión designada para el estudio de las pliegos presentados al concurso de tratamiento de basuras de Madrid. Las cifras que dan los concursantes son, para el tratamiento de 200 toneladas diarias:

	Pesetas
Instalación de un parque de incineración para 200 toneladas diarias...	10.000.000
Instalación de un parque de fermentación para 200 toneladas diarias...	6.000.000
Canon anual que debería abonar el Municipio para el sistema de incineración	995.000
Canon anual que debería abonar el Municipio para el sistema de fermentación	695.000

Tampoco entramos en detalles de técnica, por no considerarlos de este lugar ni de nuestra incumbencia.

Estos procedimientos—fermentación y cremación—están ambos sancionados por la ciencia y la experiencia y admitidos por la Sanidad oficial. La dificultad estriba en aconsejar cuál es el mejor. Desde el punto de vista sanitario puro, la incineración es incomparablemente mejor, pues si la fermentación con sus temperaturas de 70 y 80° puede verificar la asepsia del material en tratamiento, menos podrá ponerse en duda el poder, no ya de asepsia, sino de esterilización total y absoluta de la cremación con sus temperaturas de 800 a 1.000° a que alcanza, que destruyen completamente todo lo que al sanitario le estorba; pero precisamente contra esta destrucción se levanta la protesta de la técnica agrícola, que califica acertadamente de "despilfarro" y de "vandalismo químico" el acto de la destrucción total de lo que, mediante una adecuada transformación, sin contravenir los preceptos sanitarios, constituye una fuente de riqueza.

Los partidarios del sistema de fermentación condenan por este hecho el de incineración, aduciendo en pro del suyo:

a) una mayor economía de instalación y entretenimiento.

b) Completa resolución del problema sanitario.

c) Un gran beneficio a la agricultura y a la industria.

Pero como cuando se va a la solución del problema de las basuras impera siempre un criterio sanitario y la finalidad es sanitaria y no de orden agrícola, es seguramente por esto por lo que son muchos los países y las poblaciones que han adoptado la incineración. Conviene hacer constar que en alguna de ellas el resultado desde el punto de vista económico ha sido ruinoso; tal, por ejemplo, la instalación del de Colonia, cuyo Ayuntamiento, en reciente Memoria, publica que los gastos *superaban a los ingresos en 2.874.700 marcos anuales* (según el cálculo inicial del proyecto, debían ser compensados por los ingresos), y que la piedra basáltica, cuya venta estaba calculada en 1.732.00 marcos, había producido tan sólo 25.000.

Se desprende, pues, cuán difícil ha de ser, sin un estudio previo detallado y aplicado al caso particular, el decidirse por uno u otro procedimiento y la necesidad de estudiar para cada población las condiciones especiales y las circunstancias que concurren, tales como: composición de las basuras, probabilidades de venta de los subproductos obtenidos, etc.

Además, el hecho de que haya basuras unas tributarias de la fermentación y otras de la cremación, hace aconsejable en no pocos casos la instalación de un régimen mixto de cremación y fermentación, que quemaría lo inútil y aprovecharía lo utilizable por medio de la fermentación.

Además, en muchas poblaciones el montar dos instalaciones, aunque parece que implica un gasto mayor de construcción, produce una economía de transporte.

Por todas estas razones, creemos que después de estudios, informes y proyectos detallados, el Excmo. Ayuntamiento, a pesar de todo ello, no debe ir a una solución total del problema y definitivo, sino al de pequeños ensayos de los tres procedimientos descritos y aceptados (o, por lo menos.

de los dos—cremación y fermentación—) en distintos barrios de la población y de basuras de composición similar, que, al paso que una solución parcial del problema, tendría el valor de un ensayo experimental durante un período corto de pocos años, y permitiría ir luego—con un perfecto conocimiento de causa—a una solución definitiva y total.

Nuestra conclusión sería, pues, que se instalaran, por ejemplo, dos estaciones de tratamiento: una de fermentación y otra de cremación, para un tonelaje máximo de 25 ó 50 toneladas diarias, en dos barrios que pudiesen funcionar después de la misma manera definitivamente, constituyendo así para aquella demarcación una solución definitiva, y que a la vez sería un período de ensayo para la aplicación del procedimiento a todas las basuras de la capital. Aunque esta conclusión parezca demasiado simple, creemos que debería tomarse en consideración para evitar lo que ha sucedido en muchas otras poblaciones de tener que abandonar una instalación cuyo coste asciende siempre a muchos millones de pesetas, con todo y que, como es de suponer, no se lanzaron a ella sin estudios previos y cálculos precisos, como corresponden a una empresa de tal consideración.

¿El servicio debería ir a cargo del Municipio o de una Empresa? Creemos que debería ser esto último, reservándose, claro está, el Municipio el control sanitario.

CAPITULO XI

SUMARIO Y CONCLUSIONES

Durante nuestro trabajo hemos examinado un total de 8.074 ratas, de ellas 994 provenientes de Sanidad exterior, y el resto, de la captura por rateras que se hace sistemáticamente en Barcelona por el Instituto Municipal de Higiene.

De ellas hemos examinado bacteriológicamente 4.268, y entomológicamente, todas. Hemos hallado en el casco de la población un 99,9 por 100 de la especie *E. norvegicus*, y 0,1 por 100 entre *E. rattus rattus* y *E. norvegicus hibernicus*. Por lo tanto, actualmente puede considerarse *norvegicus* como la especie única prácticamente.

En cuanto a la distribución de *E. norvegicus* y *E. rattus* en relación con los focos pestosos recientes, hemos podido comprobar que la primera es la única que existe en el casco de la ciudad en la proximidad de aquéllos. En cambio, la segunda especie se encuentra limitada a la zona del puerto.

En las ratas provenientes de los barcos desratizados por la Sanidad del puerto hemos hallado la especie *E. rattus* típico y sus dos variedades *E. rattus frugivorus* y *rattus alexandrinus*.

Probablemente, dado el parecido de ambas, han sido confundidas por casi todos los que se han dedicado al estudio de las ratas de los barcos, y por ello la presencia de la primera variedad ha pasado inadvertida, a pesar de que suele presentarse en buen número.

De nuestras experiencias acerca del poder natatorio de *E. norvegicus* se deduce que, al menos en agua dulce, no puede mantenerse a flote durante más de cinco minutos, muriendo siempre ahogadas en ese tiempo; pero desde los tres minutos el animal no se preocupa de avanzar. Esto está en contraposición con los datos de los distintos autores que afirman que en bahía abierta puede nadar hasta media milla, y que en agua dulce puede atravesar ríos de la importancia del Volga.

En el laboratorio hemos experimentado acerca del poder raticida de los diversos productos empleados, y encontramos una diferencia notable a favor de la pasta fosfórica, sobre la escila, el virus de Danyz, etc.

Respecto a los ectoparásitos de las ratas, hemos hallado siete especies de pulgas, un anopluro y tres especies de ácaros, que son, respectivamente:

Afanípteros

Xenopsylla cheopis	1.985	ejemplares.
Ceratophyllus fasciatus	1.643	—
Ctenocephalides felis	973	—
Ctenocephalides canis	88	—
Ctenopsyllus segnis	265	—
Pulex irritans	35	—
Stenoponia tripectinata	3	—
TOTAL	4.992	ejemplares.

Anopluros

Polyplax spinulosus	1.649	ejemplares.

Ácaros

Echinolaelaps echidninus	1.479	—
Eulaelaps stabularis	3	—
Notoedres muris	"	—
TOTAL	1.482	ejemplares.

No ponemos los ejemplares de *Notoedres* por tratarse de un sarcóptido, que se encontraba en un porcentaje reducido de ratas.

Ahora bien, como estos parásitos han sido hallados por nosotros en distintas épocas del año, hemos segregado los datos de un año completo desde marzo de 1932 a febrero de 1933, para establecer las variaciones anuales y los porcentajes relativos durante los distintos meses del año.

Condensando las conclusiones que a este respecto hacemos en el capítulo correspondiente, encontramos que la especie predominante es el *Cer. fasciatus,* que alcanza un 47,8 por 100 anual, siguiéndole después la *X. cheopis,* con un 26,2 por 100. La curva de la *X. cheopis* muestra un paralelismo retrasado con la curva de la temperatura, debido al desarrollo larvario, y alcanza su máximo con un porcentaje de 62,2 y 74,0 por 100, respectivamente, en los meses de septiembre y octubre, mostrando su mínimo de 3 por 100 en marzo de 1932 y enero de 1933.

El *Ceratophyllus fasciatus* sigue una marcha inversa a la temperatura y tiene su máximo de 82,7 por 100 en el mes de enero, y un mínimo de 13,5 por 100 en septiembre. No se puede distinguir una correspondencia entre la curva de humedad y la de las especies de pulgas, quizá por mantenerse siempre elevada.

En cuanto a las demás especies, el *Ct. felis* sigue su marcha paralela a la de *X. cheopis,* alcanzando un máximo en agosto de 37,9 por 100; la

Ct. segnis tiene dos vértices en los meses templados, lo cual prueba quizá que necesita temperaturas moderadas, y las restantes especies se capturan tan pocas veces, que no puede sacarse de ellas conclusión alguna.

Sin embargo, la misma escasez de ***Pulex irritans*** parece indicar que sólo acude a la rata en ausencia de otro huésped más adecuado.

En cuanto al índice de pulgas por rata, es decir, a su media, nos encontramos con un índice medio anual de 0,6, es decir, francamente bajo. Si unimos a ello que la especie predominante no es la *X. cheopis,* sino el *Cer. fasciatus,* tendremos que Barcelona entra en el grupo de las poblaciones relativamente poco infectables, y cuyos brotes son prontamente combatidos.

Se han ensayado diversos métodos de cría de pulgas, entre ellos el de la placa de escayola y el de Langeron, sin obtener resultados positivos. Tan sólo por el método Leeson, y una vez corregidas las faltas de técnica, hemos podido obtener excelentes resultados y continuar durante tres generaciones consecutivas la cría de *X. cheopis* y la de *Cer. fasciatus.*

En los distintos ensayos que hemos realizado acerca de la infección experimental de virus de la seudotuberculosis de los roedores, por medio de las pulgas hemos podido comprobar que no es factible, quizá debido a una septicemia poco intensa.

Respecto a la longevidad de las pulgas, nuestros resultados han sido siempre inferiores a los obtenidos por otros autores, tanto en lo que se refiere a pulgas alimentadas diariamente como a las pulgas en ayunas. En el primer caso, la longevidad máxima la hemos obtenido con el *Cer. fasciatus,* que alcanzó unos seis meses (desde el 28-XII-32 al 20-VI-33), y en el segundo la obtuvimos con la *X. cheopis,* que permaneció durante veinticinco días sin alimentarse. Nuestros datos en este respecto se aproximan más a los que ha obtenido Leeson.

Por último, con objeto de comprobar la hipótesis de Ricardo Jorge sobre la distinta infectabilidad de las pulgas en relación con la estructura del proventrículo, hemos llevado a cabo un estudio detenido de dicho órgano, que hasta ahora no se había realizado, tanto por cortes histológicos como por disecciones, que han permitido ver bien los procesos quitinosos internos.

En éstos hemos encontrado ligeras diferencias en cuanto a su tamaño y a la forma de las quillas de que están provistos en las distintas especies; pero, sin embargo, a nuestro juicio, no son suficientes para explicar la distinta infectabilidad natural de unas y otras.

Hemos creído encontrar una explicación a este hecho, que puede servir para un futuro tema de trabajo, en el poder fagocitario de la sangre de los huéspedes habituales, pues es indudable que una pulga, cuyo animal predilecto sea resistente a la infección pestosa por esta propiedad de su sangre, ha de desembarazar de bacilos su proventrículo siempre que vuelva a nutrirse sobre él, lo cual ocurrirá, a nuestro juicio, con relativa frecuencia, coadyuvando a ello la lentitud de la digestión, que acontece cuando cambian de huésped. Esto explicaría que el *Ct. felis* y *canis,* que se encuentran sobre el perro, animal resistente a la peste, a pesar de tener el proventrículo en excelentes condiciones para el desarrollo de los gérmenes pestosos, no tenga papel importante en la transmisión de esta enfermedad, aparte, naturalmente, del poder bactericida de sus jugos digestivos.

Resultado de la autopsia en las ratas de Barcelona.—Sobre 4.268 ratas autopsiadas de julio de 1931 a enero de 1934, no hemos encontrado más que una rata sospechosa de peste (septiembre de 1931), procedente de un foco donde hubo cuatro casos de peste humana; las lesiones macro y microscópicas conducían al diagnóstico de peste, pero la inoculación al cavia fué negativa. Como dicha rata fué recogida muerta y sus vísceras estaban ya en putrefacción, la prueba de inoculación no tiene todo su valor en sentido negativo.

Una gran proporción de ratas de Barcelona, pertenecientes a *Epimys norvegicus,* son portadoras de lesiones hepáticas, producidas por un gusano nematode de la familia de los *Tricuridos* (el *Hepaticola hepática*).

En menor proporción se encuentran quistes en el hígado, producidos por el cisticerco de la *Taenia crassicolis.*

En algunos ejemplares hemos encontrado lesiones pulmonares seudotuberculosas, producidas por un germen grampositivo difteroide, el *Corynebacterium kutscheri.*

En las vísceras de las ratas recogidas muertas se encuentran frecuentemente, en los frotis, gérmenes con coloración bipolar, que pueden inducir a error con el bacilo pestoso. De ellas se aisla constantemente el colibacilo.

En las condiciones de nuestra experiencia, el colibacilo invade las vísceras una hora después de la muerte.

Es muy frecuente el parasitismo intestinal por *Himenolepis fraterna,* encontrándose este parásito en gran número en el mismo individuo.

Todos los lotes de ratas *Epimys norvegicus,* procedentes de Barcelona, y la mayor parte de las del Delta del Ebro, han demostrado ser portadoras del virus de la *espiroquetosis icterohemorrágica.*

Algunas inoculaciones practicadas con ratas *Epimys norvegicus,* procedentes de otras localidades (Reus), y las *Epimys rattus,* procedentes de Sanidad exterior (cianhidrización de buques), han dado resultado negativo en este sentido.

Los pases en el cavia del virus de espiroquetosis icterohemorrágica aislado nos han dado los resultados siguientes:

Incubación de cinco o seis días, sis grandes trastornos ostensibles.

Fiebre de 40 a 41°, que aparece al sexto o séptimo día, acompañada de enflaquecimiento, erizamiento del pelo y flaccidez muscular.

Ictericia hacia el séptimo día, aproximadamente.

Hipotermia, coincidiendo casi con la ictericia o siguiéndola de poco.

Muerte hacia el octavo o noveno día, con el cuadro típico y descrito anteriormente.

El curso de la infección puede acelerarse por una infección sobreañadida, pudiendo llegar a constituirse el cuadro típico y la muerte en cuatro días.

No todos los cavias inoculados mueren. Algunos siguen la misma marcha clínica; pero después de la icteria, curan. En toda investigación de laboratorio para diagnóstico en el hombre es prudente, pues, inocular, por lo menos, cuatro cavias, y a poder ser, con orina del enfermo de diferentes micciones.

Los cavias que han sobrevivido a una primera inoculación resisten una segunda a dosis cinco veces mayor.

Las lesiones que ofrecen a la autopsia, no siempre constituyen el cuadro típico descrito. En algunas ocasiones sólo se presenta una congestión hemorrágica difusa en los pulmones.

La rata blanca de laboratorio resiste a la inoculación por vía intraperitoneal, pero conserva el virus, puesto que, sacrificadas a los dos meses, la inoculación del triturado de sus vísceras a cobayas nuevos les provoca la espiroquetosis típica.

En las mismas zonas donde hemos capturado ratas espiroquetósicas (Delta del Ebro y Barcelona) se han dado después cuatro focos de espiroquetosis icterohemorrágica en el hombre (uno, en San Carlos de la Rápita; otro, en Jesús y María, Delta del Ebro; otro, en Hospitalet, y, últimamente, uno, en Barcelona). Los dos primeros grupos de casos se dieron en trabajadores del cultivo del arroz; el de Hospitalet, en obreros de las limpieza de acequias de regadío, y el de Barcelona ha constituído una epidemia de origen hídrico, comprobada epidemiológicamente y bacteriológicamente (aislamiento del leptospira de una fuente contaminada de la que habían bebido los enfermos).

Encuesta epidemiológica sobre la peste bubónica.—En Barcelona ha habido brotes epidémicos de peste en los años 1905, 1906, 1919, 1922, 1923, 1930 y 1931.

La epidemia de 1931 estuvo constituída por 26 casos de peste bubónica, localizada en los focos de basuras y trapos de los alrededores de la ciudad.

Las características epidemiológicas incluyen netamente este brote en las epidemias de origen murino.

Por razones de orden epidemiológico, *creemos que no hay relación de dependencia ninguna de unas epidemias con otras,* pudiéndose admitir únicamente esta relación cuando el período intermediario entre una epidemia y otra ha sido corto (1922-1923 y 1930-1931).

No es admisible, bajo ningún concepto, que haya habido desde 1905 (en que se presentó el primer brote de peste en Barcelona desde 1681) *una enzootia pestosa latente* en la población murina, manifestándose por brotes epidémicos en los años citados.

Esta conclusión nuestra se apoya en consideraciones epidemiológicas, y además, en los hechos conocidos de la extinción espontánea de la peste murina, a la que se llega por:

Resistencia o inmunidad que adquieren las ratas.

Transmisión de esta inmunidad a las nuevas generaciones.

Eliminación de los individuos menos resistentes.

Tendencia a la formación de las lesiones que han sido llamadas por la Comisión inglesa de la India *Resolving* o *Resolved plague,* que conducen a la curación.

Ausencia de transformación de esta forma *Resolving* en peste aguda o septicémica, única transmisible a los otros animales por los ectoparásitos.

Fagocitosis del bacilo en el interior del proventrículo de la pulga, ejercida por la misma sangre chupada, y aumentada cuando procede de un animal parcialmente inmunizado.

Disminución de la virulencia del bacilo en el organismo de las ratas inmunizadas.

Posible intervención de un bacteriófago antipestoso.

Desratización activa practicada por el hombre.

El origen del brote de 1931 ha sido desconocido, si bien por las razones expuestas anteriormente debe considerarse como importado, o en todo caso, que fuese importado en 1930, si se admite relación de dependencia entre la peste de estos dos años.

Relacionando el tiempo en que han tenido lugar los diferentes brotes de peste en Barcelona desde 1905, con el estudio que hemos llevado a cabo sobre los ectoparásitos, veremos que coinciden con el período de máximo desarrollo de la *Xenopsylla cheopis* (verano y otoño).

Habida cuenta de la evidente relación existente entre *ratas, basuras* y *peste,* una medida profiláctica eficaz y definitiva debe ser la solución del problema de las *basuras domiciliarias.* Como sea que un estudio técnico previo no es suficiente para el conocimiento exacto del ulterior resultado económico (según se ha demostrado con lo ocurrido en otras capitales), creemos que podría comenzarse rápidamente con la construcción de dos pequeñas instalaciones: una, de fermentación, y otra, de incineración en dos barrios distantes, que a la vez que constituiría una solución parcial, tendría el valor de un estudio experimental, que podría decidir al Municipio a adoptar para el tratamiento total el o los procedimientos que le ofrecieran más garantías de orden sanitario y económico a la vez. Las decisiones que se adoptaren no deberían prescindir de los intereses legales que ha creado el actual sistema de aprovechamiento de las basuras.

En tanto no se vaya a dicha solución, debe continuarse la desratización activa y sistemática (como se practica ahora), aun cuando no se presenten casos de peste humana ni epizootias.

TRABAJO REALIZADO EN EL LABORATORIO MUNICIPAL DE BARCELONA

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3. Case of tokelau (Tinea imbricata) of fifteen years standing in a European.

Reprinted from THE MEDICAL PRESS AND
CIRCULAR, MAY 17, 1933.

Case of Tokelau (Tinea imbricata) of Fifteen Years' Standing in a European

By Sir ALDO CASTELLANI, K.C.M.G.,
D.Sc., M.D., F.R.C.P., Director-in-Chief,
Ross Institute for Tropical Diseases

THE patient (Mr. H. A. B.) first noticed the eruption while in the Army, on active service in Arabia, in 1916. He entered the hospital at Aden ; received treatment there without any benefit. Was sent to England, and was treated in different hospitals. The condition improved, and he was sent to Bombay (1917), but before arriving there the eruption broke out violently with terrible pruritus. After a short time in Bombay he was sent back to England. Having obtained no distinct benefit from treatment in several military hospitals, he consulted, in London, Dr. Pringle (privately), who made a tentative diagnosis of tinea imbricata, and advised the patient to see me, but I was then in the Balkans. From his treatment he received some slight relief, which, however, was only temporary. The patient went before the Medical Board of India in December, 1917. No definite diagnosis was made ; he was invalided, and informed that it would be inadvisable for him to reside in tropical countries. Soon after having been discharged he went to Japan, and stayed in that country until 1922 ; also visited Manchuria and Korea in 1919–1920. He returned to England, *via* Canada, in 1922, and has not since left this country. He entered Roehampton Hospital at the beginning of February, 1932. He

benz. co.; various mercurial ointments; salicylic acid and resorcin in lanolin; betanaphthol and salicylic acid in vaseline; scarlet red; brilliant green; X-rays; ultra-violet rays.

Present Condition.—The eruption is present on most regions of the body and typical of the variety of tinea imbricata due to *Endodermophyton indicum*, the lesions being more superficial than in the type due to *E. tropicale.* The scales are fairly large, tissue paper-like and adherent, not falling spontaneously. The pattern of the eruption is best seen by examining the photographs with a magnifying lens. From the scales a fungus with all the characters of *Endodermophyton indicum* has been grown, the growth being of a pink colour, and serum very slowly liquefied. The condition is improving under treatment consisting of the application

Appearance of the eruption.

to the patches of the fuchsin-acetone-resorcin paint I introduced a few years ago in the treatment of epidermophytosis.

Tokelau (tinea imbricata), as well known, is a strictly tropical disease, being found only in certain regions of the tropics, where it is at times confused with ichthyosis—it has been called tropical ichthyosis—or with dermatitis exfoliativa; but the microscopical examination of the scales will reveal masses of fungus. When large portions of the body are affected the disease is practically incurable. A fairly complete description of the disease may be found in the "Manual of Tropical Medicine" (Castellani and Chalmers), p. 2059.

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A MON PÈRE

A LA MÉMOIRE DE MA MÈRE

A MA SŒUR

A MES FRÈRES

Témoignage de ma vive affection.

MEIS ET AMICIS

INTRODUCTION

Dans le cours de nos dernières années d'études dans cette faculté, nous avons eu l'occasion assez rare en Europe, d'observer deux foix l'éléphantiasis des Arabes. Il nous a paru intéressant de faire, à propos de ces deux cas, quelques recherches, sur l'étiologie de cette horrible et désolante affection. C'est le résumé de nos lectures que nous présentons à nos juges dans cette thèse.

La cause parasitaire, qui seule explique les observations que nous avons recueillies, est exposée d'une façon sommaire dans les dissertations inaugurales qui traitent de l'étiologie de la maladie. Ces considérations nous ont conduit à restreindre notre sujet aux rapports de l'éléphantiasis avec la filaire, et nous avons pensé qu'ainsi limité, il pouvait recevoir un développement plus complet. Nous nous proposons de donner un aperçu de l'histoire de ce parasite, d'analyser les travaux relatifs à son rôle dans la genèse de l'affection qui nous occupe, et de discuter les objections qui ont été formulées contre son influence.

Nous diviserons notre étude en trois chapitres. Le chapitre I sera consacré à l'histoire naturelle de la filaire, il comprend l'historique de sa découverte, ses caractères anatomiques, la périodicité de son apparition dans les capillaires cutanés, ses migrations et sa métamorphose.

Ces notions sont indispensables pour comprendre le mode d'action du ver.

Dans le chapitre II, nous étudierons le rôle pathogénique de la filaire et nous exposerons les arguments et les faits sur lesquels il repose.

Enfin, dans le troisième chapitre, nous examinerons les critiques qui ont été faites à la théorie parasitaire.

Mais avant d'aborder notre sujet, nous sommes heureux de présenter à nos maîtres le témoignage de notre profonde reconnaissance. Que MM. les Professeurs G. Sée, Peter et Potain dont nous avons pu apprécier la grande bienveillance daigne accepter l'hommage de l'admiration que nous ont inspirée leurs savantes et intéressantes leçons. Nous prions M. le professeur Bouardel, doyen de cette faculté, de recevoir nos remerciements pour l'honneur qu'il nous fait en acceptant la présidence de notre thèse.

CHAPITRE I

De la filaire du sang de l'homme

A. — *Historique.*

En août 1863, Demarquay découvrit, dans le liquide laiteux provenant d'une hydrocèle de la tunique vagériale d'un jeune homme de la Havane, l'œuf et l'embryon d'un ver particulier. Il les décrivit la même année comme appartenant à un helminthe nématode, n° Davaine, d'après un dessin qui lui fut présenté, déclara que ce parasite « ne ressemblait par sa forme à rien de décrit (1) ».

Ce fait, malgré son intérêt et son importance, tomba dans l'oubli, sans doute à cause de son isolement et de l'impossibité d'un contrôle immédiat.

Aussi, quand, le 4 août 1866, le naturaliste allemand du nom de Wucherer, en cherchant la Bilharzia hœmatobia dans le sang d'un chylurique du Brésil, rencontra le même eutozaaire il crut pouvoir le considérer comme nouveau dans la science (2).

La découverte de l'illustre chirurgien avait passé inaperçue ; celle du naturaliste, publiée seulement en 1868 à une époque où lui ou ceux qui en avaient connaissance avaient déjà réuni de nombreux cas semblables, fixa davantage l'attention. Ainsi s'explique pourquoi cette découverte dont l'honneur revient en réalité à Demarquay est généralement attribuée à Wucherer, et aussi pourquoi les observateurs ne se sont mis à la recherche du parasite qu'à partir de cette dernière année.

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(1) Demarquay. — *Gazette médicale de Paris*, XVIII, 1863.

(2) Wucherer. — *Gaz. méd. da Bahia*, 1868 ; trad. in Arch. de méd. navale 1870, t. III, p. 141.

Bientôt, en effet, Leuckart (1) le signala dans un caillot sanguin qu'il avait reçu de Bahia ; Lewis (2), dans l'Inde, et Salisbury (3), aux Etats-Unis, l'observèrent dans l'urine de plusieurs individus atteints de chylurie tropicale. En 1870, Crevaux (4), chez un malade de la Guadeloupe, et Spencer Cobbold (5) chez un autre de Port-Natal le retrouvèrent dans les mêmes conditions que leurs devanciers.

En 1872, Lewis (6) le vit, non plus dans l'urine où on l'avait presque exclusivement rencontré jusqu'alors, mais dans le sang, chez un hindou qui souffrait de diarrhée chronique. Le ver vivait dans ce liquide saguin comme dans son élément naturel, le médecin de Calcutta le considéra comme un véritable hématozoaire et le baptisa du nom de *filaria sanguinis hominis* qui lui a été conservé (7).

La découverte de la filaire dans le sang éclaira de la plus vive lumière ses relations pathologiques. Lewis lui-même en recueillit les fruits deux ans plus tard.

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(1) Leuckart. — Die parasiten der Menschen.

(2) Lewis. — Ann. report on the sanit... in India, 1868.

(3) Salisbury. — Brit. med. Journal, 28 juil. 1872.

(4) Crevaux. — Hématurie chyl. et grais. Th. Paris, 1872.

(5) Cobbold, — Brit. méd. Journ. 1872, 29 juillet, Méd. Times.

(6) Th. Lewis. — On a hœmatozoon, etc. Calcutta 1872. Analyse in Journ. de l'anat. et de la physiol., 1873.

(1) Lewis. — On a hœmatozoon, etc. Calcutta 1874. Analyse in Arch. de méd. nav. t. XXX, 1878.

B. — Des rapports de la filaire du sang avec l'éléphantiasis des Arabes.

Poursuivant ses recherches dans la voie qu'il avait ouverte, ce médecin ne tarda pas à signaler la présenée de l'animal dans une autre affection ; il le trouva dans le sang et dans la lymphe d'un grand nombre d'individus atteints d'éléphantiasis des Arabes. En face de ce fait et de sa fréquence, il n'hésita pas à admettre une relation de cause à effet entre le parasite et la maladie (1).

L'année suivante, un savant et perspicace observateur dont nous retrouverons souvent le nom dans le cours de ce travail, Patrick Manson, médeccin des dounnes chinoises d'Amoy, se mit à la recherche de l'hématozoaire. Il le rencontra chez un dizième des habitants de cette ville. Les uns étaient en parfaite santé, d'autres étaient atteints de maladies qui évidemment ne pouvaient être rapportées au ver, mais le plus grand nombre souffraient d'éléphantiasis et de chylurie. Il attribua ces deux affections à la présence de l'eutozoaire, et posa les bases de la théorie qui porte aujourd'hui son nom.

Cependant, la filaire adulte restait toujours inconnue. Ce n'est que le 21 décembre 1876 que Baucroft (2) la découvrit à Brisbane (Australie). Il l'a trouva d'abord dans un abcès lymphatique du bras, puis peu de temps après, dans une hydrocèle du cordon spermatique. Au mois d'août 1877, Lewis démontra sa présence dans un cas d'éléphantiasis (1). Nous rapportons cette intéressante observation.

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(1) Lewis. — The pathological significance. Calcutta 1874 et the Laucet, 1875.

(2) Baucroft. — Cité par Cobbold in the Laucet, 14 juil. 1877.

Le malade, un jeune Bengalais, était atteint de tuméfaction variqueuse du scrotum. Il n'avait jamais eu ni chylurie, ni lymphorrhagie cutanée. La peau de ses bourses était parsemée de vésicules et présentait la consistance molle et spongieuse ordinairement observée en pareil cas. On procéda à l'ablation de la tumeur. Dés la première incision, il s'écoula un liquide jaunâtre, séro-lactescent qui coagula rapidement. Le microscope permit d'y voir de nombreux embryons de filaire, que l'on retrouva dans toute l'épaisseur des tissus, dans les varices lymphatiques et dans une hydrocèle du cordon spermatique. Mais, dans toutes ces parties, il fut impossible de découvrir le progéniteur. On allait renoncer à sa recherche quand l'attention fut attirée par un peloton de filaments blanchâtre contenu dans un caillot sanguin ; c'était le ver parent.

Silva Araujo et Victorino Pereira l'ont retrouvé, quelques mois après, dans un autre cas de lympho-scrotum, compliqué de chylurie et de Craw-Craw (2).

Les exemplaires rencontrés par ces différents observateurs étaient des femelles. Le connaissance du mâle ne date que de 1886 ; nous la devons à deux médecins brésiliens, Figueira de Saboia qui en a trouvé un specimen dans le ventricule droit du cœur (1) et Pedro S. de Magalhaes qui l'a décrit (2). En 1888, Sibtharpe, professeur au *Médical College* de Madras, a recueilli les deux sexes, étroitement unis, à la surface de section d'une tumeur lymphangiectasique du scrotum (3).

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(1) Lewis. — The Laucet, II, 1877 et 1882. Arch. méd. nav. t. XXIX, p. 200.

(2) S. Araujo. — *Gaz. méd. da Bahia*, 1877. Arch. med. nav. 1878.

C. — Caractères anatomiques de la filaire du sang humain.

Malgré le petit nombre de vers adultes rencontrés jusqu'à présent, nous possédons, grâce aux travaux de Lewis, de Mauson, de Sp. Cobbold, de Magalhaes et de Bourne *(4)* une description assez complète de la filaire du sang. (loc. cit.*)*

Nous rappellerons brièvement ses caractères anatomiques.

La filaire est vivipare ; Lewis et Manson ont vu sortir de ses tubes utérins des embryons absolument identiques à ceux qu'ils ont trouves dans le sang et dans la lymphe. Les œufs qui sont en liberté dans la circulation sont des produits d'avortement ; ils sont en tout semblables à ceux qui sont encore contenus dans les ovisacs et présentent dès leur ponte un commencement de segmentation. Ils sont de forme ovale, dépourvus de coque et recouverts seulement d'un chorion. Leur contenu est granuleux et séparé de la paroi par un espace clair. Au moment de leur expulsion, ils mesurent 21 *u* sur 18 *u* ; *in utéro*, quand l'embryon y devient visible, ils ont atteint les dimensions de 30 *u* sur 37 *u*.

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(1) F. de Saboia. — Cité par le suivant.

(2) Magalhaes. — *Revue des Cours de la Fac. de Rio-de-J.*, t. III, 1886.

(3) Sibthorpe. — Brit. méd. journal, t. I, p. 1344, 1889.

(4) Bourne. — Ibidem t. 1, p. 1050, 1888.

Les embryons fourmillent dans la circulation ; leur nombre dans la masse totale du sang a été évalué à 140.000 (Lewis), à 1.500.000 (Myers), à 2 millions (Manson). Ce sont de petits vers arrondis, allongés, effilés à une extrémité, obtus à l'autre, mesurant de 125 à 300 *u* en longueur et de 7 à 11 en largeur. Ils sont dépourvus de tout indice de tubes digestif et d'organes reproducteurs. Leur corps est d'une transparence extrême ; il est parfois enveloppé d'une gaîne qui est également d'une translucidité parfaite. Cet étui, auquel la théorie parasitaire fait jouer un rôle considérable, n'est pas élastique, il ne suit pas le corps de l'animal dans ses contractions et forme tantôt à une extrémité tantôt à l'autre, un cul de sac allongé qui simule une sorte de fouet.

La filaire embryonnaire est animé de mouvements ondulatoires qui, joints à sa forme, l'ont fait comparer à un petit serpent.

L'état adulte n'a guère été rencontré qu'au cours d'opérations ayant pour but l'ablation d'un lympho-scrotum ou l'amputation d'un membre éléphantiasé.

La femelle est de teinte opaline. Elle mesure de 8 à 10 centimètres. Son diamètre transversal va en augmentant de l'extrémité orale, ou il est de 0 $^{m}/_{m}$ 28, au tiers antérieur du corps où il atteint 0 $^{m}/_{m}$ 70 ; puis diminue jusqu'à l'extrémité caudale qui se termine par une pointe mousse (Magalhâes). Ce ver est comparé tantôt à un cheveu ou à crin, tantôt à « un mince fil de catgut qui serait animé et frétillant ». Il est recouvert d'une cuticule assez épaisse. Sa partie antérieure arrondie et claviforme est supportée par un col grêle. Sa bouche est terminale, circulaire et dépourvue de valves. L'anus est situé à la face ventrale, au sommet d'une pupille, à peu de distance de l'extrémité de la queue. Le vagin court et bifurqué, s'ouvre près de la terminaison céphalique. A part un tube digestif étroit, la cavité centrale est presque exclusivement réservée à l'appareil génital constitué par deux longs ovaires remplis d'œufs et d'embryons de tous les âges.

Le mâle est également de coloration opaline, mais il est plus mince que la femelle. Sa largeur est 0 $^{m}/^{m}$ 40 au tiers anttérieur du corps et de 0 $^{m}/^{m}$ 1 à l'extrémité postérieure ; sa longueur est de 8 centimètres environ. La partie céphalique est arrondie et se continue sans ligne de démarcation avec le reste du corps. La bouche est terminale, circulaire et inerme ; la queue est mousse, enroulée en spirale et présente à sa face ventrale huit paires de papilles, situées, en nombre égal, en avant et en arrière du cloaque. Cet orifice s'ouvre à une dizaine de millimètre de la terminaison postérieure et donne passage à deux spicules long, arqués et inégaux. Un œsophage muni d'un puissant bulbe pharyngien. Un intestin d'abord dilaté puis rectiligne, un rectum étroit et court, un appareil génital constitué par un tube étroit et sinueux complètent l'anatomie de la filaire mâle.

D. — *Périodité des migrations de la filaire dans l'organisme humain.*

La filaire a échappé à un nombre d'observateurs et non des moins habiles, par suite de l'ignorance où ils se trouvaient de la périodicité de ces migrations dans l'organisation. Aussi, croyons-nous utile de dire quelques mots de ce curieux phénomène dont nous devons la connaissance à Manson.

Ce médecin, frappé de l'abondance des embryons à certains moments de la journée et de leur rareté ou même de leur absence à d'autres chez les mêmes sujets, s'est astreint à examiner toutes les trois heures le sang de ses malades. Il a constaté ainsi que si les filaires disparaissent ordinairement pendant le jour des capillaires périphériques, ils y retournent vers 6 ou 7 heures du soir avec une « exactitude presque militaire » pour employer l'expression de Cobbold. Leur nombre s'accroît à mesure que la nuit s'avance, et, vers minuit il atteint son maximum ; on peut alors en compter une centaine dans une seule goutte de sang. Puis ils deviennent de moins en moins abondants, et 9 ou 10 heures du matin, ils ont le plus souvent complètement disparu (1).

Que deviennent-ils pendant le jour ? Les autopsies qui seules auraient pu nous le dire, font défaut. On en est donc réduit aux hypothèses. Deux sont en présence. Myers pense que les vers succombent après avoir circulé quelque temps dans le sang, et qu'ils sont remplacés le lendemain par un nouvel essaim, produit d'une ponte nouvelle. Il croit sa manière de voir suffisamment justifiée par la différence de vivacité qu'il a observée entre les embryons du matin et ceux du soir. Du reste, ajoute-t-il, la circulation ne tarderait pas à être entravée si elle n'était débarassée de ses hôtes à mesure qu'ils se multiplient (1). Mauson, au contraire, suppose que ceux-ci se réfugient pendant le jour dans quelque organe profond pour se reposer. Son opinion est conforme à ce que nous savons de l'histoire de la *filaria immitis* du chien, qui présente la plus grande analogie avec celle de l'homme. Les jeunes de ce ver, bien qu'ils ne disparaissent jamais complètement de la circulation, s'y montrent néanmoins périodiquement plus nombreuv la nuit que le jour. En faisant l'autopsie de chiens foudroyés par l'acide prussique ou la strychinne, Manson s'est assuré que les embryons s'accumulent pendant le jour dans les gros troncs vasculaires du thorax et de l'abdomen. De plus, le fait signalé par Carter (2), à savoir qu'on peut les rencontrer chez l'homme pendant les heures de veille, et qu'ils sont même en plus grande abondance durant les accès fébriles diurnes que la nuit, milite contre l'hypothèse de Myers.

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(1) Manson. On the periodicity, etc. in China Cust. Gaz. n° 22. 1881.

A quelle loi naturelle obéit ce phénomène de périodicité ? on ne sait. Peut-être n'est-elle liée qu'aux alternatives de sommeil et de veille de l'hôte humain. En effet, on peut changer les heures des repas et toutes les autres habitudes des individus filariés sans modifier aucunement l'ordre de l'apparition du parasite ; mais que l'on vienne à les tenir éveillés la nuit et à les faire dormir le jour ; on observe une interversion correspondante dans la périodicité du ver. (Manson, Mackensie (1).

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(1) Myers. China Cust. Gaz., n° 21, 1881 et n° 32, 1886.

(2) Carter. The Laucet I, 1882.

E. — Métamorphose de la filaire du sang humain.

L'œuf ou l'èmbryon des échinorshynques, des ténias, du ver de Guinée, de la trichine, de l'oxyure, doit passer dans un autre organisme pour que le cycle vital de l'espèce puisse s'accomplir. C'est une loi presque absolue dans l'histoire des entozoaires, la *filaria sanguinis hominis* n'y fait pas exception. Son hôte intermédiaire est un moustique. Beaucoup d'espèces peuvent l'héberger pour un temps plus ou moins long, une seule, le *culex mosquito* l'amène jusqu'aux phases ultimes de son développement larvaire. Ce moustique est un diptère, nocturne comme le ver, et très commun dans certains pays tropicaux où on le voit voltiger près du plafond des chambres au ment du coucher du soleil. Il est reconnaissable aux caractères suivants : plus petits que les autres variétés avec lesquelles on le rencontre, il est de couleur brun foncé. Sa patte postérieure est inerme, mais recouverte d'appendices délicats disposés à la façon des barbes d'une plume. Son rostre se termine par une bulbe conique qui porte à sa base deux longues antennes inégales, velues et moniliformes. Ses ailes présentent des côtes, et des appendices semblables à ceux des pattes frangent leur périphérie (Myers, loc. cit.)

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(1) Mackensic. The Lancet II, 1881, p. 398 et 707.

Le Culex mâle est incapable de perforer les téguments, mais la femelle possède un appareil buccal assez puissant pour les transpercer. Elle est d'une voracité telle que deux minutes de succion lui suffisent pour se gorger de sang. Avec les globules, elle aspire le ver, et, fait curieux, elle semble même avoir une affinité particulière pour ce dernier : le nombre des embryons que l'on retrouve dans son corps est proportionnellement bien supérieur à celui que renferme le sang de l'individu qu'elle a piqué. (Lewis (1), Sonsino (2), Manson).

Son repas achevé, le moustique va se fixer au bord de l'eau, choisissant de préférence une eau stagnante. Là. il digère la nourriture qu'il vient de prendre et mûrit ses œufs. Au bout de quatre à cinq jours, il est arrivé au terme de son existence ; il s'abat sur l'eau, y dépose ses œufs et meurt (3).

Pendant ce laps de temps, la filaire a poursuivi sa métamorphose, qui est achevée du 6^e au 7^e jour. Elle se montre alors pourvue d'un tube digestif complet, d'une bouche fermée par quatre papilles, d'un anus situé en avant de la queue et entouré de trois ou quatre papilles étalées à la façon des pétales d'une fleur. Elle s'est dépouillée de son enveloppe ; sa longueur est de 1^{mm}, 58, sa largeur est 0^{mm}, 3. Le contact de l'eau qui paraissait lui être nuisible dans le cours de ses transformations la vivifie maintenant et active ses mouvements. Elle dévore le cadavre de l'insecte ou bien celui-ci est détruit par la décomposition qui ne tarde pas à s'en emparer, et la larve se trouve en liberté dans l'eau, désormais organisée pour une vie indépendante (Manson).

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(1) Lewis, 14 th. Annual report of the sanitary. etc, in India.

(2) Sousino. Med. Times. and. Gaz. 13 et 22 mai 1883.

(3) Mauson-China Cust. Gaz. sept. 1877. Arch. Med. Nar. 1884. t. XLII p. 321. .

C'est dans ce nouveau milieu qu'elle semble avoir été retrouvée par d'autres observateurs. En effet Magalhàes dans l'eau potable de la Carioca à Rio-de-Janeiro (1), Moura de Brazil (2) dans celle du jardin batanique de cette ville et Sp. Cobbald dans celle de certains puits d'Egypte (3) ont recueillie des filaires qui présentaient la plus grande ressemblance avec celle de l'homme.

Il est donc probable que le parasite s'introduit dans l'organisme humain avec l'eau de boisson, et qu'il s'ouvre une voie à travers les parois du tube digestif jusqu'au point où il va élire définitivement domicile.

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(1) Magalhâes. — O Progresso medico de Rio, 1877 — Arch. de med. Nar.
t. XXIX p. 313.

(2) M. Brazil. Ibidem. p. 314.

(3) Cobbold. Brit. mèd. Journ. sept. 1882. Th. de Lorentz, Par. 1890 N° 96.

CHAPITRE II

Du rôle pathogénique de la filaire dans l'éléphantiasis des Arabes.

A. — *Exposé de la théorie parasitaire.*

En 1874, Lewis découvrit l'embryon de la filaire dans le sang, dans la lymphe et dans l'urine de plus de 30 individus, presque tous étaient atteints d'éléphantiasis ou de chylurie. Devant une association aussi fréquente, il n'hésita pas à avancer les propositions suivantes :

1° Ces affections sont dues à la présence de l'hématozaaire ;

2° Elles résultent de la gêne mécanique qu'il apporte à la circulation des fluides nutritifs dans les capilaires et les lymphatiques (loc. cit.).

Pour expliquer le rôle pathologique du parasite, il s'appuya sur ce qu'il avait observé chez les chiens de Calcutta qui sont infestés par des vers très voisins de ceux de l'homme, et admit, par analogie, que les troubles circulatoires déterminés par l'entozoaire humain, reconnaissent l'une ou l'autre des lésions suivantes :

a) Des tumeurs, formées par l'animal adulte ankysté le long des vaisseaux sanguins et lymphatiques, entravent la circulation par la compression qu'elles exercent, soit directement sur ces vaisseaux soit indirectement sur es nerfs de la partie intéressée ;

b) Les vers embryonnaires, ou plutôt en voie de développement, perforent, dans leurs migrations actives, les tissus nerveux et vasculaires et produisent des lésions plus ou moins permanentes ;

c) Ou bien, libres dans les capillaires, ils peuvent par leur extrême mobilité et leur accumulation soit active soit accidentelle, former des embolies qui amènent la rupture des parois délicates de ces vaisseaux.

Lewis, avec une merveileuse intuition. avait trouvé la pathogénie de la maladie éléphantoïde. Mais l'analogie qu'il avait supposée entre le mode d'action des filaires du chien et celle de l'homme n'était pas fondée. Ni les tumeurs vermineuses, ni l'entassement embolique des embryons n'ont été constatés chez ce dernier. Quand à la perforation des tissus, elle est bien difficile à comprendre de la part de vers qui sont dépourvus de tout appareil buccal et de tout mouvement de progression.

Aussi, quand Manson reprit cette hypothèse, l'année suivante, il esseya de l'asseoir sur des faits observés directement chez l'homme, sans pourtant négliger les enseignements que lui fournissait la pathologie comparée. Il admit que l'éléphantiasis résulte d'une stase mécanique produite par la présence de l'entozoaire dans les tissus ; mais il donna au mode d'action du parasite une explication bien différente de celle proposée par son confrère de l'Inde.

L'embryon, qui était à cette époque (1875) le seul état sous lequel la filaire fut connue, avait été rencontré dans le sang, dans la lymphe et dans les varices lymphatiques. Il était donc propable qu'il vivait dans la circulation, au moins habituellement. Sa présence et surtout celle des œufs témoignaient de l'existence du progéniteur en quelque point de l'organisme. Mais où ? sans doute dans le système lymphatique, puisque ses embryons été trouvés dans la lymphe alors même qu'ils étaiens absents du sang ; plus précisément, dans un vaisseau lymphatique. puisque ses dimensions, à en juger par celles de l'œuf, ne lui permettaient pas de pénétrer dans les ganglions. De là, il était naturel d'inférer que le ver-parent verse ses produits de conception dans le courant de la lymphe qui les porte jusqu'aux ganglions : que ses jeunes, grâce à leur petitesse et à leurs mouvements vigoureux, passent dans les lymphatiques efférents et finalement dans le sang.

La filaire adulte devait, dans certains cas, obstruer le vaisseau dans lequel elle est logée soit directement par son volume, soit indirectement par l'inflammation qu'elle occasionne. Elle vit ordinairement en parfaite harmonie son hôte, mais qu'elle puisse devenir nuisible à un moment donné, nul doute, ne peut exister. Presque tous les entozoaires donnent lieu à des troubles, les filaires du chien, en particulier, qui sont proches parentes de celles de l'homme, déterminent parfois les désordres les plus graves (1).

Telle est la suite de raisonnements sur lesquels Mauson édifia la théorie de la cause parasitaire de l'éléphantiasis des Arabes et des autres lymphoses tropicales. Au mode d'action multiple supposée par Lewis, il substitua un mécanisme unique : l'obstacle siège toujours en dedans des vaisseaux et ces vaisseaux sont toujours des lymphatiques.

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(1) Mauson. Observations on lympho-scrotum. Méd. Times, 15 novembre 1875.

Il avait d'abord admis que l'obstruation est le fait du ver-parent ; plus tard, il reconnut que le rôle prépondérant, mais non exclusif, doit être accordé aux œufs et surtout aux embryons morts sans avoir pu se débarrasser de leur chorion. Les uns et les autres sont, en raison de leur volume et de leur pasivité, arrêtés par la première barrière ganglionnaire qu'il rencontrent, et, là, ils interceptent la circulation (1).

L'obstruction est établie, elle est plus ou moins complète. Ses conséquences immédiates sont la stase de la lymphe et l'augmentation de sa tension.

Si les voies lymphatiques ne sont que partiellement fermées, il en résulte un état variqueux des ganglions et des vaisseaux afférents ; mais, grâce aux anatomoses, la circulation continue, bien que génée et charrie les filaires dans le sang. Le lympho-scrotum, la chylurie, les varicosités des glandes lymphatiques avec embryons dans le sang, tels sont les symptômes de ce degrès d'obstruation.

Si l'obstruction est complète, deux cas se présentent :

a) Ou les lymphatiques se rompent et il s'établit une lymphorrhagie plus ou moins permanente. Dans ces conditions, la lymphe ne stagne pas complètement, elle circule bien qu'en sens rétrograde et reste fluide. Cette forme d'obstruction se traduit par la lymphorrhagie du scrotum ou de la jambe, par les varices des ganglions avec présence des vers dans ces glandes et peut être dans la lymphe épanchée, mais non dans le sang ;

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(1) Manson. Additionnal notes, etc., in Custo, Gaz., 1879, n° 18, p. 31.

b) Ou bien les lymphatiques ne se rompent pas ; il y a alors stase absolue dans les vaisseaux situés en amont des ganglions. L'induration et l'hypertrophie de ces glandes et des tissus en résultent, par conséquent, l'éléphantiasis. Aucun ver ne peut être trouvé dans le sang, car, pas un d'eux n'a pu franchir le crible ganglionnaire oblitéré. Le progéniteur meurt, pour ainsi dire, étouffé par la lymphe qui s'organise et la masse de ses jeunes. Dans l'eléphantiasis vrai aucun embryon ne peut donc être observé ni dans le sang ni dans la lymphe, si ce n'est au début de l'affection.

Ainsi, du degré de l'obstruction, de la rupture ou de la résistance des lymphatiques dépendent la forme de la maladie éléphantoïde et la présence ou l'absence du parasite dans le sang et dans la lymphe (1).

Cependant, tous les sujets filariés ne sont pas voués fatalement à l'éléphantiasis ou autres états morbides attribués à l'hématozoaire. L'apparition d'une quelconque des manifestations de la filariose est liée au nombre d'œufs que pond le ver, à la fréquence de ses avortements, au degré de vitalité des embryons, à leur nombre et enfin à la nature des tissus intéressés. La maladie est un accident dans l'histoire de la filaire, mais un accident, à la vérité, tres fréquent (2).

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(1) Manson. Transact. of the path. soc, of. London, 1881, t. 32, p. 285.

(2) Manson. Cust. Gaz. méd. rep. 1877, n° 14.

Quant au siège de l'éléphantiasis, il est déterminé par la position qu'occupe la filaire mère et des autres conditions que nous venons d'énoncer. « Supposons, dit Manson, le ver parent logé dans un lymphatique de la jambe droite et voyons ce qui arrive. Ses œufs et ses embryons sont charriés d'abord dans les ganglions inguino-fumoraux droits qu'ils obstruent, puis, par la voie anastonomique, à travers le scrotom, jusqu'aux glandes correspondantes du côté opposé qui sont oblitérées à leur tour. La stase peut être alors complète dans les deux membres et dans le scrotum. Les vaisseaux de cette dernière partie, qui ont donné passage à la lympe provenant des jambes en même temps qu'à celle qu'ils conduisent normalement, sont, par le fait de ce surcroît de travail, distendus et variqueux. S'ils se rompent, il y a lympho-scrotum, et, dans le liquide qui s'écoule périodiquement, on trouve des embryons et des œufs. Comme la lymphe n'a pas traversé de gànglions, elle est claire, couleur paille et pauvre en cellules. Si, au contraire, les lymphatiques des bourses, bien soutenus qu'ils sont par le dartos, ne cèdent pas, mais résistent, la lymphe s'accumule dans les endroits où l'action de la pesanteur se fait le plus sentir, à une jambe ou aux deux et au scrotum, comme c'est souvent le cas. Ces parties augmentent de volume, et la circulation, peu active ou nulle, entraîne la mort du progéniteur. »

« La présence de celui-ci dans l'épaisseur du scrotum amène les mêmes résultats. »

« Mais s'il est dans un tronc lymphatique du bassin ou de la région lombaire, la stase est déterminée par les œufs et les embryons qui envahissent les lymphatiques des reins, des uretères ou de la vessie. Il peut en résulter de la chylurie. Comme, dans ce cas particulier, le parasite se trouve probablement entre deux ganglions et que les anastomoses du vaisseau dans lequel il est logé sont gardées par des ganglions, si toutes ces glandes viennent à être obstruées, il peut se faire qu'il soit impossible aux produits de conception de passer dans le système sanguin. Généralement cependant, grâce à la richesse des anastomoses dans ces régions, l'obstruction n'est pas complète et le ver-parent survit.

« Que l'obstruction pelvienne ou lombaire soit étendue et qu'elle siège à gauche, par exemple, la lymphe régurgite par la voie anastomotique interne ou externe d'abord jusqu'aux ganglions inguinaux du même côté, puis, jusqu'à ceux du côté opposé dont les conduits sont peut-être perméables. La pression est, par ce fait accrue dans les lymphatiques des jambes et du scrotum ; d'où tendance à l'éléphantiasis ou au lympho-scrotum. S'il en résulte un lympho-scrotum, comme la lymphe épanchée a traversé de nombreux ganglions dont quelques-uns peuvent communiquer avec le tube alimentaire, elle est chyleuse ou même sanguinolente et trés riche en cellules.

« Une fois que la stase est permanente dans une région ou que la lymphorrhagie est établie, les lymphatiques contenus dans la zone primitive de l'obstruction sont soulagés, car, la quantité de lymphe qu'ils ont à transmettre est moindre que celle qui provenait auparavant du siège de développement de la maladie. Aussi, il est d'observation que l'ablation d'un lympho-scrotum est souvent suivie d'éléphantiasis, la lymphorrhagie joue le rôle de soupape de sûreté. »

En un mot, l'éléphantianis siégeant presque toujours aux jambes ou au scrotum, l'obstacle est situé tantôt en amont, tantôt en aval des ganglions inguinaux. Dans le premier cas, la lymphe est fluide et claire ; dans le second, elle est dense, blanchâtre, chyleuse.

Cette théorie est généralement adoptée par les partisans de l'origine parasitaire de l'éléphantiasis des Arabes. Elle est, on peut déjà le prévoir, solidement construite. Les faits les plus indiscutables sont venus, depuis le petit nombre d'années qu'elle compte, la confirmer dans tous ses points principaux.

A. — *Fréquente association de la filaire avec l'éléphantiasis des Arabes.*

L'éléphantiasis des Arabes est une maladie très anciennement connue, Galieu l'a décrit dans sa *Méthode thérapeutique à Glaucon* et l'a nettement séparé de la lèpre (1). Cependant la forme de cette affection qui s'accompagne de varices lymphatiques et de lymphorrhagie cutanée (éléphantiasis nœvoïde) et qui frappe si souvent le scrotum (lympho-scrotum) était assez mal étudiée jusqu'à ces derniers temps. Sa première description ne date que de 1854 ; nous la devons à Ardaseer Jamsetjee qui en rapporte un cas typique (2).

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(2) Jamsetjee, cité par Mauson in Med. Times and Gaz., 13 nov. 1875.

(1) Barallier. Dict. de Jaccoud, art. éléphantiasis.

« Le malade, un robuste marchand parse de 50 ans, souffrait depuis 17 ans et demi d'une tuméfaction du scrotum qui, disait-il, s'était montrée à la suite d'un traumatisme. La peau de ses bourses était rugueuse et épaisse ; elle présentait à la partie inférieure et antérieure de petites vésicules d'où s'écoulait, parfois en très abondance, un liquide blanc laiteux. Le flux apparaissait irrégulièrement et quand il s'arrêtait les vésicules s'affaissaient. Son existence, pensait l'auteur, empêchait la tumeur scrotale de s'accroître. »

Cet état est presque toujours associé à la présence de la filaire. En effet, Manson a montré en 1875, que sur 13 individus qui en étaient atteints 10 présentaient ce parasite (1). Plus tard, grâce à l'habileté qu'il acquit en cherchant les filaires du chien, ce médecin a pu trouver celle de l'homme dans tous les cas de varices lymphatiques des bourses (2). G.-C. Roy est arrivé à des résultats analogues, il a vu l'entozoaire douze fois dans douze cas de cette affection (3). Sibthorpe nous dit qu'il lui a presque toujours été possible de le rencontrer dans la forme de la maladie éléphantiaque que caractérise l'exsudation de la lymphe qu'elle siégeàt aux jambes ou au scrotum (4). En mars 1877, F. dos Santos a montré la présence du ver dans le sang d'une tumeur de cette nature (5) ; il l'a revu quelques mois plus tard chez deux autres sujets dans la lymphe qui s'écoulait de leurs jambes (1). En octobre de la même année, Magalhâes l'a reconnu dans l'épaisseur du scrotum d'un individu opéré pour éléphantiasis variqueux (2). Peu de temps après, il l'a aperçu de nouveau dans le sang et dans la lymphe d'une femme atteinte d'éléphantias de la lèvre (8). Déjà, au mois d'août, Lewis avait découvert la filaire adulte et sexuée dans une tumeur lymphangiectasique des bourses (3). Silva Araujo et Victorino Pereira l'ont retrouvée bientôt après dans les mêmes conditions. Enfin, en 1888, Sibthorpe a recueilli le màle et la femelle, étroitement unis, à la surface de section d'un lympho-scrotum (4).

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(1) Manson. China Cust. med. reports, sept. 1877, n° 14.
(2) Manson. China Cust. med. reports, sept, 1878, n° 18, p. 31.
(3) G.-C. Roy. The Lancet I, 15 avril 1882.
(4) Sibthorpe. Brit. med. Journal, v. I, p. 1344. 1880.
(5) F. dos Santos. Gaz. med. da Bahia, mars 1877.

L'éléphantiasis nœvoïde est donc si souvent lié à la présence du ver, qu'il peut-être regardé comme le type des manifestations filariennes. Sa nature parasitaire est indiscutable.

Cette conclusion était-elle applicable à l'éléphantiasis ordinaire, à cet état d'hypertrophie des membres ou du scrotum qui n'est accompagné ni de varices lymphatiques ni de lymphorrhagie cutanée ni de chylurie? Les partisans du parasitisme l'affirment. L'éléphantiasis vrai, disent-ils, est aussi souvent associé à la filaire que le lympho-scrotum. Ce qui a pu faire penser le contraire c'est que les médecins ont rarement l'occasion de l'observer à son début, les malades se souciant peu d'un gonflement qu'ils croient passager. Or, quand le mal est avancé, le ver-parent est déjà mort, étouffé par la lymphe qui s'est organisé en tissu solide, et ses jeunes, qui avaient pu passer dans la circulation générale à la faveur d'une obstruction d'abord incomplète, n'étant pas renouvelés ont également disparu. Mauson, particulièrement, s'en est assuré en cherchant ceux-ci dès l'apparition de l'affection. En 1875, il les a trouvés cinq fois chez 25 individus qui étaient atteints d'éléphantiasis pur du scrotum ou des jambes, soit dans la proportion de 20 0/0 ; tandis que sur 607 autres individus qui présentaient l'apparence de la santé ou qui souffraient de maladies indifférentes, il ne les a rencontrés que 26 fois, soit donc une proportion de 4,28 0/0. Hall et Paterson nous ont appris que le parasite n'existe que 8,66 des habitants de Babia, il n'infeste qu'un dixième de la population d'Amoy. Du reste, l'éléphantiasis ordinaire et le lympho-scrotum sont une seule et même maladie, ils reconnaissent par conséquent la même cause, la filaire. L'identité de ces deux affections, niée par MM. T. Fox et Farquahr, repose sur des arguments d'une grande valeur que nous allons exposer.

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(1) F. dos Santos. Cité par J. de Moura. Th. de Concours. Rio, 1877.

Id. Analyse in Arch, med. nav., 1878.

(2) Magalhâes. Arch, de med. nar, t. XXIX, p. 208, note.

(3) Lewis. The Laucet, II, 1877 et 1881. Arch. nar., XXIX, p. 200...

(4) Sibthorpe. Loc. cit.

B. — Identité de l'éléphantiasis ordinaire avec le lympho-scrotum.

La connexion de ces deux états pathologiques a été notée pour la première fois, en 1869, par le docteur Carter (1). En 1866, Sir J. Fayrer a montré que les lésions anatomique sont les mêmes dans les deux affections. Il a décrit un cas d'éléphantiasis nævoïde du scrotum dans lequel la tumeur enlevée pesait plus d'une livre et présentait tant à l'œil nu qu'au microscope tout les caractères de l'éléphantiasis commun (2). Mais c'est surtout à Manson que nous devons d'avoir établi l'identité de ces deux états morbides. Ils constituent, dit-il, la même maladie sous des formes légèrement différentes ou à des stades successifs de son évolution. En effet, ils atteignent la même partie, le scrotum ; ils sont également accompagnés d'inflammation locale et d'acces fébriles intermittents et irréguliers ; ils débutent l'un et l'autre par les lymphatiques ; leur distribution géographique, autant qu'elle est connue, est la même, ou du moins, le lympho-scrotum n'a été rencontré que dans les pays où l'éléphantiasis est endémique ; enfin, on les voit souvent se réunir ou se succéder chez le même individu.

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(1) Carter. Transact. of. med and physic. Soc. of Bombay, 1861, 1862.

Tous ces points sont généralement admis, sauf un seul, l'origine lymphatique de la maladie (1). Nous en parlerons à propos de l'anatomie pathologique. Quant à la coexistence du lympo-scrotum et de l'éléphantiasis ordinaire et à leur succession chez le même sujet, elles sont établies par des observations aussi nombreuses qu'intéressantes. En voici quelques exemples :

1er cas. — Un malade d'Allan-Webb souffrait depuis 10 mois d'une tuméfaction du scrotum, accompagnée d'écoulement d'un liquide visqueux et blanchâtre. Le flux s'étant arrêté brusquement, le scrotum doubla bientôt de volume et prit l'aspect éléphantiaque (2).

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(2) Fayrer. Clinical Surgery in India, 1866.

(1) Mauson. — Obs. on lympho-scrotum, etc. in Med. Times, 13 nov. 1875.

2ᵉ cas. — Le maladé présentait à la partie inférieure des bourses un petit orifice qui, pendant la marche,livrait passage à un mince filet de lymphe. Le scrotum fut enlevé à cause de son volume, le malade guérit et quitta l'hôpital. Mais il rentra bientôt avec tous le signes d'éléphancie de la jambe droite. Son sang-éontenait des filaires (Mauson).

3ᵉ cas. — Les bourses exsudaient une fois par mois, un liquide blanchâtre. L'écoulement n'ayant pas reparu pendant quelque temps les parties affectées grossirent graduellement et prirent l'aspect ordinaire de l'éléphantiasis,maladie qut nécessita une opération 7 ans plus tard. La tumeur enlevée présenta tous les caractères des tissus éléphantiasés (du même auteur).

4ᵉ cas. — Exemple typique de lympho-scrotum. La tumeur excisée, sa surface de section laissa voir une peau d'un demi pouce d'épaisseur doublée intérieurement d'une substance gélatineuse, éléphantiasis (du même).

5ᵉ cas. — Un autre malade du même auteur était atteint d'une tuméfaction du scrotum. La peau de la partie supérieure de ces bourses était couvertes de nombreuses vésicules qui fournirent à la ponction, 10 onces d'un liquide couleur paille. Les téguments du pénis étaient tellement épaissis que le gland était complètement invisible, éléphantiasis

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2. Allon-Webb. — Indian Annals of med science, 1855.

La légère différence qui existe entre les deux formes de la maladie est expliquée par la théorie parasitaire. Dans le lympho-scrotum, l'obstruction n'est que partielle. la circulation continue, bien qu'elle soit entravée, l'augmentation de tension de la lymphe distend les vaisseaux et les rend variqueux. Dans l'éléphantasis commum, la stase est absolue, la lympbe s'organise en tissu conjonctif imparfait, d'où l'hypertrophie de la partie intéressée. L'obstruction, d'abord partielle, peut devenir complète par suite des pontes répétées de la filaire ou par l'extension de l'inflammation, le second état vient compliquer le premier.

A ceux qui regardent la combinaison de ces affections comme fortuite on peut répondre qu'elle est très fréquente et qu'elle se montre presque toujours dans le même ordre, d'abord varices lymphatiques, puis éléphantiasis.

C. — Identité de l'éléphantiasis des Arabes et de la chylurie tropicale

L'identité de ces deux affections a été, en raison de leur fréquente coïncidence, soupçonnée dès 1866 par J. Fayrer (1). En 1874, Lewis a admis qu'elles scnt dues l'une et l'autre à la gène de la circulation que la filaire détermine dans les capillaires et les lymphatiques. La même année, il a apporté de nouveaux faits tendant à prouver cette affinité étiologigue. Carter, Beale, Sousino, Roberts et les auteurs brésiliens ont également noté la fréquente coïncidence de l'éléphantiasis et de la chylurie. Ils regardent ces deux états comme des manifestations peu différentes entre elles de l'infection filarienne.

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(1) Fayrer. *The Laucet*, 16 et 30 mars 1878.

Le parasite logé dans les lymphatiques des voies urinaires y détermine des varices analogues à celles que l'on observe sur la peau du scrotum. Si celles-ci se rompent, la lymphe passe dans l'urine. A la vérité, l'occasion ne s'est pas encore présentée de vérifier ces lésions à l'autopsie, mais on est conduit à admettre leur existence si on considère :

1° Qu'elles sont communément observées dans d'autres parties du corps ;

2° Qu'elles sont une conséquence naturelle du mode d'action reconnu au ver ;

3° Que l'urine dans ce cas présente les mêmes caractéres et les mêmes réactions que son mélange artificiel ou chyle ;

4° Qu'à la disparition du chyle de l'urine succède souvent une lymphorrhagie cutanée et, *vice versà*, que l'arrêt de l'écoulement cutané est suivi de la réapparition de la coloration blanchâtre de l'urine.

Nous ne pouvons nous empècher de citer quelques exemples relatifs à la combinaison, à la succession et à l'alternance des deux états qui nous occupent en ce moment.

1er cas. — Un malade de Carter souffrait depuis quatre mois d'un lympho-scrotum. L'écoulement laiteux apparaissait irrégulièrement et sans cause appréciable, puis s'arrêtait. Chaque fois qu'il cessait l'urine devenait chyleuse et coagulable en même temps que les ganglions inguinaux diminuaient de volume. (Loc. cit.)

2e Cas. — Un malade de Roberts présentait à la partie tnférieure du scrotum de nombreuses vésicules remplies d'un liquide blanc laiteux. Trois fois au cours de cette affection son urine devint chyleuse. Dès que la chylurie était établie les vésicules s'affaissaient au point de disparaître complètement.

3e Cas. — Un malade de Mauson était atteint de la même affection scrotale. Le liquide extrait par piqûre des délations variqueuses s'échappait d'abord claire, puis laiteux, et enfin sanguinolent. L'urine de cet individu éttait chyleuse depuis une semaine, après t'issue du fluide des bourse elle devint normale.

4e Cas. — Un autre malade du même auteur souffrait depui trais ans d'un lympho-scrotum bien caractérisé. A son admission à l'hôpital ses bourses exsudaient une abondante quantité de liquide chyleux. Au bout d'une semaine il appela l'attention sur son urine qui, disait-il, n'avait pu passer pendant 8 à 10 heures. Elle était abondante, d'une coloration blanc rougeâtre et coagulait rapidement. Le scrotum fut excisé. Le lendemain de l'opération l'urine devint aussi blanche que du lait et atteignit 20 onces. Le malade rentra chez lui. mais. deux mois après, il revint à l'hôpital, son urine continuait à être blanche, bien qu'elle reprit parfois son aspect normal. Une petite portion d'un pouce carré du scrotum montrait des vésicules, elle fut excisée. Depuis cette époque, cet individu urina de grandes quantités de liquide chyleux, 70 à 80 onces par jour.

Puisque ces trois états pathologiques, chylurie, lympho-scrotum et éléphantiasis ordinaire, si disparates au premier abord, sont une seule et même maladie, ils dépendent par conséquent de la même cause. Or, les deux premiers sont presque constamment associés à la filaire.

D. — *Présence des œufs et des embryons de la filaire dans les ganglions.*

Le plus souvent, dit la théorie parasitaire, l'obstruction d'où résulte la maladie éléphantiaque, est produite par les œufs ou plutôt par les embryons, qui n'ont pu se débarrasser de leur chariot. Ils sont arrêtés dans les détroits ganglionnaires et là, ils interceptent la circulation.

Manson a constaté, en 1879, que dans presque tous les cas d'éléphantiasis, une petite quantité de lymphe peut être extraite des ganglions hypertropluës au moyen d'une seringue de Pravaz. Cette lymphe contient, soit épars, soit réunis en petites masses, des corps qui ressemblent les uns à des étuis de filaires abandonnés et affaisés, les autres à des débris d'œufs avortés. Il a trouvé ces corps dans tous les cas d'éléphantiasis pur, non variqueux, non lymphorrhagique, qu'il a eu l'occasion d'examiner.

De plus, dans un groupe de neuf cas, montrant la combinaison des deux formes de la maladie, ou, plus exactement, le début de la période hypertropluque, il a découvert, par le même procédé, des embryons moribonds. Ces vers présentaient une extrême lenteur dans leurs mouvements, un aspect ridé dénotant une existence ancienne. Il n'y en avait aucun dans le sang (1).

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(1) Manson. Additionnel notes on fil. sang. hom. in Cust. gaz. méd. reports, 1879, n° 18, p. 31.

Ce second ordre de faits nu peut être interprêté que comme il suit. L'obstruction complète, à en juger par le commencement de l'augmentation de volume des parties affectées, ne remontait qu'à peu de temps, les embryons de la filaire n'avaient pas encore succombé, mais leur état de vieillesse prouvaient que de nouveaux essaims n'avaient pas été versés depuis longtemps dans la circulation. Il était donc probable que le progéniteur était mort et que ses jeunes, ne pouvant se reproduire sans passer dans l'organisme d'un autre animal, ne tarderaient pas à avoir le même sort (Siegfried). Les cas avancés de la maladie nous moutrent qu'il en est ainsi ; quand l'obstruction est ancienne, on ne trouve plus que les restes quelquefois méconnaissables du ver.

Myers, bien qu'il n'admette pas le rôle généralement reconnu au parasite, témoigne de sa présence dans les ganglions, alors même qu'il est absent du sang (loc. cit.).

E. — Présence de la filaire adulte dans un vaisseau lympathique.

L'obstruction est, plus rarement il est vrai, le fait du ver parent.

En 1880, un individu atteint d'éléphantiasis nævoïde du scrotum vint trouver Manson. L'examen de son sang ne révéla aucun embryon de filaire, mais, en revanche, dans le liquide aqueux et coagulable qui s'épanchait de ses bourses on en rencontra un grand nombre.

Ce médecin diagnostiqua une obstruction des lymphatiques siégeant en amont des ganglions. Il appuyait sa manière de voir sur les considérations suivantes :

1° La lymphe qui a traversé les ganglions est laiteuse ou même sanguinolente et très riche en cellules ; celle qui s'écoulait du scrutum de ce malade, était limpide et fluide comme lorsqu'elle provient des radicules lymphatiques ;

2° Les ganglions n'étaient pas variqueux comme ils l'eussent été si l'obstacle avait siégé en aval ;

3° La présence d'embryons dans la lymphe et non dans le sang prouvait que l'obstruction était complète.

Il était donc probable que le ver adulte avait son siège dans le scrotum et que par l'opération on arriverait à le découvrir. En effet, la tumeur enlevée, on vit s'agiter sur la surface de coupe un ver du diamètre d'un crin de cheval. Il faisait saillie d'un vaisseau lymphatique dilaté dans lequel il était encore partiellement contenu. L'autre extrémité du vaisseau sectionné était visible à la surface de la plaie et laissait sourdre une gouttelette de lymphe quand on exerçait une pression sur les ganglions de l'aîne (1).

.La pièce qui montrait la filaire *in sitû*, fut présentée en 1881, à la Société de médecine de Londres par le docteur Thin.

Ce fait unique jusqu'ici suffit à démontrer que le parasite adulte à son siège dans un vaisseau lymphatique.

F. — Anatomie pathologique. origine lymphatique de l'éléphantiasis.

L'éléphantiasis des Arabes est une maladie du système lymphatique. Il débute ordinairement par un frisson et une douleur très vive siégeant en un point de la région qui doit être envahie, aîne, face interne de la cuisse, scrotum, testicule. Bientôt la fièvre s'allume, et la peau se marbrent de plaques rougeâtres et diffuses d'où partent des traînées qui suivent le trajet des lymphatiques. Les ganglions sont tuméfiés et douloureux, le membre est augmenté de volume comme œdématié (lymphangite). Au bout de deux à trois jours la fièvre tombe, la rougeur et la chaleur de la peau disparaissent. La tuméfaction ganglionnaire persiste et ne s'efface jamais complètement. Après un intervalle qui varie, chez le même sujet, d'un mois à un, deux et trois ans, les mêmes phénomènes fébriles et inflammatoires reparaissent quoique avec moins d'intensité. Ces paroxysmes laissent le membre chaque fois plus tuméfié ; ils marquent toute l'évolution de la maladie.

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(1) Mauson. Lympho-scrotum shoVing fil. in sitû. Transact. of the poth. Soc. of London 1881, *t*, 32, p. 285.

Les caractères du liquide épanché, l'aspect des vaisseaux dilatés qui le contiennent avant son évacuation, la sensation si caractéristique communiquée au doigt qui palpe les ganglions toujours hypertrophiés suffisent à établir l'origine lymphatique du lympho-scrotum. Pour l'éléphantiasis ordinaire, le fait est moins évident, mais non moins réel. Il est admis par la majorité des médecins qui ont eu souvent l'occasion d'observer cet état pathologique sous les tropiques, tels sont Aslard (1), Mohamed-

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(1) **Alard.** *Hist. d'une maladie part. au vysh. lymph.* Paris, 1808.
— *De l'inflammation dds vaisseaux absorbants.* Paris, 1824.

Ali-Bey (2), Fayrer (3), Carter (4), Roberts (5), Beale (5), Sonsino (6) et les auteurs brésiliens. Paget (7) a noté que l'éléphantiasis coexiste avec un état de dilatation et d'obstruction des lymphatiques et que ces vaisseaux contiennent dans quelque cas un fluide laiteux semblable au chyle. James Hendy (8), dans une jambe amputée pour cette affection, a trouvé les vaisseaux à sang blanc dilatés au point d'admettre une grosse plume dans leur lumière et tellement amincis et friables qu'ils ne pouvaient recevoir une injection de mercure sans se rompre. Manson les a vus dans un état analogue au cours d'une opération, le bout cardiaque de l'un deux avait acquis un calibre tel qu'il jugea utile d'y poser une ligature. Dans le cas où cet auteur a trouvé la filaire adulte, le lymphatique qui la contenait était manifestement lésé. T. Fox et Farquhar, bien qu'ils rejettent la nature parasitaire de la maladie, reconnaissent qu'elle résulte d'une obstruction des lymphrtiques qui sont dilatés en certains points, imperméables en d'autres (9) Bentley a observé les mêmes lésions et les rapporte à la filaire (10). Schlitz (11),

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(2) Ali-Bey. *De l'éléph. des Arabes*. Par. 1860.

(3) Fayrer. *Loc. cit.*

(4) Carter. *Loc, cit.*

(5) Roberts, Beale. Cités par Mauzon.

(6) Sousino. *Med. Times and Gaz.* 1883.

(7) Paget. Lectures ou Surgical Pathology. London.

(8) J. Heudy. *Traité de la maladie gland, des Barbades*. Traduct. — Alard in. *Mémoires de la Soc. d'Emulation*, t. IV, p. 44.

(9) T. Fox et Farquhar. On certain eudemic skin, etc. London 1879.

(10) Beutley. *The Laucet*, 1er juin 1878.

(11) Schlitz. Cité par Boiteux, *in Rev. de Chirurg*, 10 fév. 1882.

Bœkel (1), Rindfleisch (2), Guibout (3), Cornil (4) ont noté également l'augmentation de volume des lymphatiques. Ce dernier auteur a trouvé les ganglions chroniquement enflammé (5). « La condition instrumentale de l'affection, dit M. Besnier (6) est toujours une irritation et une obstruction du système lymphatico-veineux d'une région du corps. » Pour M. Brocq le mal est consécutif à des irritations répétées du système lymphatique, irritations dont dont l'agent peut être la filaire (7). Enfin M. Guibout regarde comme sa cause principale tout ce qui peut apporter une gêne mécanique à la circulation soit sanguine soit lymphatique.

Mais peut-on trouver de meilleures preuves de l'origine lymphatique de l'éléphantiasis que les faits que nous avons déjà rapportés et qui montrent la coexistence, la succssion et l'alternance de la chylurie, de la chylodermie avec cette affection.

Les lésions observées dans les vaisseaux et les ganglions lymphatiques sont-elles l'œuvre de la filaire ? Il est difficile de le nier quand on se rappelle que celle-ci a été vue dans ces vaisseaux et dans ces glandes, alors qu'elle était absente partout ailleurs. Elle est un corps étranger libre dans la circulation. Pourquoi lui refuser le rôle d'agent obstruant, irritant et phlegmasique reconnu à tout autre corps étranger placé dans les mêmes conditions, à l'embolie sanguine par exemple ?

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(1) Bœkel. Cité par Carmichaël. Th. Paris, 1889-90.

(2) Rindfleisch. *Ibidem.*

(3) Guibout. Leçons sur l'éléph. des Arabes. *Gaz. des hôp.*, 1877, p. 810.

(4) Cornil. *Hist. path.*

(5) — *Progrès médical*, 11 sept. 1883.

(6) Besnier. Lecons sur l'éléph. des Arabes. *Gaz. des Hôp.*, 1878, n° 128.

(7) Brocq. *Traitement des maladies de la peau*, Par. 1890.

Mais supposons l'obstruation produite. Comme conséquences, le cours de la lymphe est gêné ou même arrêté ; la tension de ce liquide est accrue dans les vaisseaux situés en amont de l'obstacle. Le fait physiologique de la transsudation d'une certaine quantité de sérosité à travers les parois des capillaires est exagéré. Le liquide épanché devient considérable, il infiltre le tissu connectif. L'obstacle apporté à la circulation en retour par les lymphatiques s'oppose à sa réception, l'œdème est établi. Cette hydropisie, reconnaissant une chose qui persiste ou se renouvelle est chrpnique d'emblée. L'exsudat s'organise ; aidé par les éléments préexistants et les parties qui avoisinent le siège de l'obstruction, il aboutit à la formation d'un tissu conjonctif imparfait. C'est ce tissu de nouvelle formation qui, sur les coupes de tumeurs éléphantiaques, prend l'aspect d'une substance gélatineuse. Cependant les générations successives d'œufs et d'embryons viennent de temps en temps provoquer de nouvelles poussées inflammatoires qui activent le processus néoplasique. Finalement, la région acquiert cette énorme et monstrueuse augmentation de volume qui a valu son nom à la maladie. « Lorsque la circulation est gênée, dit M. Guibaut, ils se produit de l'œdème ; cet œdème est la conséquence de l'épanchement en dehors des vaisseaux d'un liquide qui devait rester en dedans de ces vaisseaux. Sous l'influence de poussées inflammatoires répétées ou d'une inflammation chronique permanente, ce liquide s'épaissit, se charge d'éléments cellulaires qui s'agglomèrent, s'organisent et constituent un néoplasme, un tissu nouveau. Ce tissu s'applique sur les parties constituantes d'un membre, aponévroses os, il fait corps avec tous ces organes, il les épaissit, les dénature, les hypertrophie et, permettez-moi ce mot les éléphantiasise. » (loc. cit.).

G. — Distribution géographique identique de la filaire, du moustique et de l'éléphantiasis des Arabes.

En Afrique, la filaire a été observée en Egypte par Sousino (1), Fayrer (2), Cobbold (3) et Cauvet (4) ; à la Réunion par Foucervines (5) ; à Maurice et à Madagascar, par Nativel (6), et Giraud (7).

En Asie, Lewis, Manson et Sibthorpe nous ont montré combien elle est commune aux Indes et en Chine. Elle n'existe pas dans l'île de Formose, mais au Japon, elle a été rencontrée à Kiou-Chiou, Goto, Hirado-Chima, Amakousa, par Scheube (8), à Nagasaki, par Torikala (9), d'Oïta et Beukema, à Hondo et à Tokio, par Bœlz (10).

Bancroft l'a trouvée en Australie (1), Chassaniol et Guyot, à Taïti (2).

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(1) Sousino, cité par le suivant.
(2) Fayrex. Fil. œgyptiaca. The Laucet, 26 août 1876.
(3) Cobbold. Loc. cit.
(4) Cauvet. Arch. de méd. navale, t. XXVI, p. 360.
(5) Foucervines. Cité par Ch. Robin, leçons sur les humeurs, p. 844.
(6) Nativel. De la chylurie intertropicale, th. Par. 1886.
(7) Giraud. Etude sur la fil. de Wucherer, th. Bord. n. 37, 1886.
(8) Schenbe. Prog. méd. 1884, p. 605.
(9) Torikala. Cité par R. Blanchard, in tr. de zool. méd. Paris, 1890.
(10) Bœlz. Ibiden.
(1) Baucroft. The Laucet I, 1885, p. 380.
(2) Chas. et Guyot. Arch. de méd. nar. t. 29, p. 61.

En Amérique, elle a été vue dans le sud des Etats-Unis, par Salisbury (3) et Guitéréas (4) ; à Saint-Thomas, par Ponttoppidan (5), à la Guadeloupe, par Crevaux, Maurel (6), et Papin (7); à Cuba (observ. de M. L Audain (8) ; à la Guyane, par Winckel (9) ; à Buénos-Ayres par Wernicke (10). Nous savons combien elle est répandue au Brésil (Hall (11), Paterson, S. Lima, F. des Santos, S. Araujo, P. S. de Magalphaès, F. de Saboia, loc. cit.

L'éléphantiasis est endémique dans tous ces pays. Son aire de distribution est, il est vrai, plus étendue que celle du parasite ; elle comprend toute la zone située entre le 30e degré de latitude nord et le 35e de latitude sud dans les deux hémisphères. Cette différence s'explique par les difficultés qu'offre la découverte du ver. Mais la rapidité avec laquelle l'aire de celui-ci s'est agrandie depuis quelques années permet de prévoir qu'elle atteindra bientôt les mêmes limites que celle de la maladie.

Le moustique abonde dans les mêmes localités que la filaire et la maladie. Lewis l'a signalé dans l'Inde, Manson en Chine, Sonsino en Egypte. Silva Araujo au Brésil, etc. Ces médecins ont pu suivre plus ou moins complètement les métamorphoses de l'embryon dans son corps. Où le culex mosquito est absent, l'affection est très rare ou même inconnue. En effet, Myers, sur 15.000 malades reçus à l'hôpital de Formose, n'a observé que trois cas d'éléphantiasis : les trois individus qui en étaient atteints venaient du continent, Amoy et ses environs. où le mal règne avec une frequence telle, que un dizième de la population en est affecté. Recherchant la cause de l'absence de l'éléphancie dans l'île, Myers s'est assuré que le moustique n'y existe pas (1). Un autre fait éclaire d'une vive lumière le rôle pathologique de la filaire, c'est l'importation de la maladie à la Barbade, où elle était inconnue au siècle dernier (Hendy). Il y avait dans ce pays, fait remarquer M. B. Roncière (2) deux facteurs, des moustiques et des hommes, le troisième, l'entozoaire, faisait défaut. Un individu l'y a apporté un jour et l'affection s'y est rapidement répandue, elle est maintenant endémique.

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(3) Salisbury. loc. cit.
(4) Guitéréas. Med. News Philadelph. 1886.
(5) Poutoppidan. Cité par Nouvenoux, th. Par., 1884.
(6) Maurel. Tr. des malad. palud. à la Guyane.
(7) Papin, Hématurie eud. des p. chauds, th. Bordeaux 1886.
(8) LAudain. Observation in th de Lorentz, Par. 1890, n° 96.
(9) Winckel. Cité par M. R. Blanchard.
(10) Wernick. Ibidem.
(11) Loc. cit.

Si l'éléphantiasis était indépendant de l'hématozoaire ce serait une coïncidence bien étrange que celle qui nous montre la maladie, le parasite et son hôte réunis dans les mêmes localités. « Pour battre réellement en brèche la théorie de Mauson, il faudrait, dit M. Brassac, démontrer que l'éléphantiasis est complètement inconnu dans des régions où existent la filaire et le moustique qui lui sert d'hôtes pour ses métamorphoses (1) ». Or, des faits de ce genre font absolument défaut. Nous voyons, au contraire, le désordre glandulaire des Barbades endémique dans les mêmes lieux, que deux autres maladies propres aux pays chauds, et dont la nature parasitaire est généralement admise, telles sont la dracontiase produite par le ver de Guinée et l'hématurie du cap due à la bilharzia hœmatobia.

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(1) Myers. Loc. cit.

(2) B. Roncière.. Arch, méd. nav. XXX, 1878.

(1) Brassac. Dict. encycl. des sc. nied,, art. éléphantiasis.

D'autres arguments en faveur de la cause animée ont été tirés de l'efficacité de certains médicaments, essences, copahu, térébenthine et préparations mercurielles, dont le pouvoir parasiticide est bien connu. C'est ainsi que Bentley, guidé dans ses recherches sur la thérapeuthique de l'affection par les connaissances étiologiques nouvelles, a empleyé avec le plus grand succès les préparations hydrargyriques. Il est à désirer que ce mode de traitement se vulgarise, que les médecins des pays chauds, encouragés par les bons résultats qu'il a déjà fournis, lui accordent plus de faveur qu'il n'en a rencontré jusqu'à présent.

Résumé des arguments et des faits qui militent en faveur de la théorie parasitaire.

1° La filaire est, sinon constamment, du moins presque toujours, associée à l'éléphantiasis des Arabes, incontestablement plus souvent qu'à l'état de santé ou de maladies indifférentes.

2° Ses œufs et ses embryons sont communément trouvés dans la lymphe et dans les ganglions, au milieu des tissus affectés, alors même qu'ils sont absents du sang et partout ailleurs.

2° Le ver adulte a été découvert directement dans un lymphatique, et ce vaisseau était manifestement lesé.

4° L'éléphantiasis est la même maladie que la chylurie, dont l'origine filarienne est généralement admise.

5° Il débute par le système lymphatique ; ses lésions primitives sont celles de ce système, comme le démontre l'anatomie pathologique.

6° La pathogénie qu'on lui reconnaît généralement est une gêne de la circulation lymphatique.

7° Il est endémique partout où la filaire existe, et celle-ci n'a jamais été rencontrée dans une localité où il est inconnu. Il n'a pas été observé, du moins endémiquement dans les localités où le moustique, intermédiaire nécessaire entre l'entozaire et l'homme, fait défaut.

8° Les médicaments parasiticides ont une influence marquée dans sa thérapeutique.

9° Presque tous les entozoaires sont nuisibles. Les filaires du chien, la trichine, le ver de Médiné qui, particulièrement, sont de la même famille que la filaire du sang humain, deviennent, dans de certaines conditions, la cause de désordres les plus graves.

Peut-on refuser de voir dans les rapports de l'hématozoaire de l'homme avec l'éléphantiasis des Arabes une relation de cause à effet ? Qu'il nous soit permis, avant de répondre, de passer rapidement en revue les autres causes auxquelles on attribue une influence prépondérante ou même exclusive dans l'étiologie de cette maladie.

H. — Comparaison de la cause parasitaire de l'éléphantiasis des Arabes avec celles auxquelles on accorde une influence exclusive.

Les autres causes qui suffiraient à expliquer l'endémicité de l'éléphantiasis des Arabes dans les pays chauds sont le paludisme (Waring (1), Clot-Bey (2), Lallement (3),

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(1) Waring. Statistique in th. de Clarac Par. 1881.

(2) Clot-Bey. Elephantiasis des Arabes, n° 34.398 et Gaz. des hôp. n^os 80 et 85.

(3) Lallement. Cité par Clarac.

Claudio da Silva (4) et Vinson) (5), la sypilis (Larrey père (6) etc), la scrofule (Caradec) (7), et les irritations répétées de la peau (Levacher) (8).

L'étiologie paludéenne paraît assez bien établie ; on connait nombre de cas où aucune autre influence ne peut être incriminée. M. Besnier, parmi plusieurs autres auteurs, a rapporté, en 1878, trois cas de ce genre. Cependant quelques médecins ont voulu accorder à cette cause un rôle prépondérant ou même exclusif. Ils font reposer leur manière de voir sur l'existence encore douteuse des lymphangistes palustres, sur la ressemblance des accès intermittents avec la fièvre éléphantoïde, sur l'existence de l'engorgement ganglionnaire et sphénique dans les deux affections, enfin sur l'endémicité de l'éléphantiasis dans les foyers palustres.

Il reste encore à prouver que les lymphangites des pays chauds sont d'origine miasmatique. Mazaé Azéma (1) nous a appris que les fièvres telluriques n'ont fait leur apparition à Bourbon qu'en 1868, tandis que la lymphangite endémique était connu de tout temps. Il ne semble pas, dit-il, que celle-ci ait été influencée dans sa marche et dans sa fréquence, par les conditions nouvelles que la malaria a créées. Quelques heures de séjour dans le pays uffisent à l'immigrant européen pour contracter les fièvres tandis que les angioleucites ne l'atteignent qu'après un acclimatement de 5 années environ.

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(4) Cl. da Silva. Thèse de Rio, 1884.

(5) Vinson. Contrib. à l'étude de la lymph. grave. Arch. méd. nav. 1876.

(6) Larrey. Relation chir. de l'Exp. d'Egypte. Paris 1803.

(7) Caradec. Mémoire à l'académie de médecine, 1879.

(8) Levacher. Guide médical aux Antilles.

(1) M. Azéma. Consid. prat. sur l'éléph. des Ar. Gaz. méd. de Paris, 1858.

Les accès éléphantiaques diffèrent des accès palustres par leur irrégularité constante, par leur durée et la longueur des intervalles qui les séparent. Si l'engorgement des ganglions est caractéristique du désordre des Barbades il n'en est pas de même de la tuméfaction de la rate. En 1872, Mauson (1) écrivait : « nos idées sur la pathologie de la maladie sont celles généralement admises, à savoir que c'est une affection des lympathiques déterminée par des influences palustres. Cependant nous n'y avons jamais ou que très rarement observé l'augmention de volume de la rate ». Il faudrait pour accorder une influence exclusive à l'impaludisme, qu'il se montrât partout où l'éléphantiasis est endémique. Or, il n'en est pas ainsi, la coendémicité des deux affections est bien souvent en défaut. Hillier n'a jamais vu l'éléphantiasis chez les soldats européens sur qui la malaria sévissait dans l'Inde. Les indigènes des pays chauds possèdent une immunité presque absolue contre l'inection masmiatique et pourtant ils sont les plus éprouvés par l'éléphantiasis. Les immigrants de race caucasique, au contraire, deviennent d'autant plus sujet à être atteint par ce dernier mal, qu'ils ont acquis une plus grande résistance contre le premier.

L'éléphantiasis est commun dans certaines iles de l'Océanie, la Nouvelle-Calédonie et Taïti, dans certaines localités de l'Inde, Pondichéry par exemple, où la malaria est rare ou même complètement inconnue. Il se montre au contraire à l'état d'exception pathologique dans d'autres lieux ou sévissent, avec la plus haute intensité, les fièvres à quinquina (1). A Cochin, une rangée de 16 cottages qui étaient ravagée par ces fièvres, étaient absolument indemnes de toutes manifestation éléphantiasique. L'éléphantiasis est si commun aux iles Samoa que la population masculine en est atteinte dans la proportion de 50 0[0 ; les indigènes vous apprennent que le mal se montre surtout aux voisinages des étangs ; ces iles réunissent toutes les conditions favorables à la genèse du miasme paludéen, cependant on n'y a jamais observé la malaria (2). Celle-ci est endémique dans plusieurs contrées de la zone tempérée, telles sont la Bresse et la Polohne, les marais Pontins et la campagne romaine, la Grèce, les bouches du Danube, l'éléphantiasis ne se montre dans ces localités que par cas isolés et rares. Maintenant que le parasite de l'infection palustre est connu on peut demander des preuves plus évidentes que celles qui ont été apportées jusqu'ici pour soutenir son influence.

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(1) Mauson. China med. rep. mars 1872.

D'autres auteurs se basant sur ce que un grand nombre d'individus atteints d'éléphantiasis sont entachés de syphilis, ont accordé à cette dernière affection la plus large part dans l'étiologie de la première. Qu'une maladie qui frappe si profondément que la syphilis le système lymphatique, qui détermine si souvent des ulcérations cutanées pouvant devenir le point de départ de lymphangites;

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(1) Dr A. Collas. Cité par M. Corre. Traité clin. des maladies des p. chauds. Par., in-8°.

(2) Koniger. Cité par H. Barth. Annales de dermatologie, 1891.

qu'une telle maladie ait une certaine influence sur le développement de l'éléphantiasis, il est rationnel de l'admettre. Mais à considérer combien la première de ces affections est fréquente dans les grands centres de population des climats tempérés et combien l'autre y est rare, il est permis d'affirmer que cette influence doit être bien restreintes sous les tropiques.

Le même argument peut être invoque contre la scrofule. Son action doit être très limitée.

Quant aux causes irritatives, elles sont aussi nombreuses que frèquentes dans les pays chauds, où les indigènes ont l'habitude de marcher nu-pieds et nu-jambes. Levacher (loc. cit.) leur reconnaît le rôle prédominant dans la genèse de l'éléphantiasis ; et ils semblent, en effet, aider puissamment à son développement. Cependant, leur influence a été également exagérée. Dans deux localités voisines. qui ont les mêmes mœurs et les mêmes coutumes, qui sont soumises aux mêmes conditions climatériques, où l'on observe les mêmes dermatoses, dans l'une l'affection est endémique, dans l'autre elle est très rare ou même inconnue. Cette inégalité de fréquence, suivant les lieux, s'explique au contraire parfaitement par la cause animée. Le moustique filarien fourmille à Anoy, il n'existe pas à Formose qui est situé à deux pas du continent ; comme conséquence, l'éléphantiasis n'est observé dans l'île que chez les individus qui viennent du continent (loc. cit.).

L'influence attribuée à la chaleur, à l'eau, à l'humidité, au voisinage des étangs et des marais, revient en réalité à la filaire. Nous savons que ce parasite ne vit que dans les zônes tropicales et circatropicales, que le moustique, son propagateur, affectionne les lieux humides, qu'il choisit les eaux stagnantes pour y déposer ses œufs et mourir ; que ce sont ces eaux qui, bues sans précaution, transportent l'entozoaire dans l'organisme.

Les faits rapportés à l'hérédité par Virchow, Duchassaing, Godard, Azéma et Waring trouvent une explication plus exacte dans la théorie parasitaire. Tous les membres d'une famille, boivent le même eau et absorbent le ver. Mais de ce que cette pseudo-hérédité ne se montre pas dans toutes les familles habitant la même ville, on ne peut tirer un argument contre la cause parasitaire de l'éléphantiasis. La maladie n'est pas une conséquence fatale de la présence de l'entazoaire dans l'organisme, mais un accident.

Quant à la contagion, si elle était prouvée,elle serait contraire à la cause animée ; il faut que le ver passe dans l'organisme d'un autre animal avant de pouvoir se reproduire chez l'homme. Les médecins qui ont admis ce mode de propagation sont ceux qui ont confondus l'éléphantiasis des Arabes avec la lèpre. Cette dernière affection est réellement contagieuse, sa nature bacillaire est solidement établie. Pour la première, au contraire, qu'elle que soit la cause qu'on lui reconnaisse rien n'explique la transmission directe de l'homme à l'homme. Du reste, des médecins qui l'ont souvent observée aux Antilles aucun, à la connaissance de M. Brassac, n'admet sa contagiosité. Le peuple lui-même, toujours enclin à accepter les préjugés de ce genre, n'y croit pas. Loin de séquestrer les éléphantiaques, on les garde comme domestiques dans tous les détails de la vie intime, l'homme continue à vivre avec sa femme devenue éléphantiaque ; les mères n'hésitent pas à allaiter leurs enfants.

En somme, tout en reconnaissant une certaine influence à quelques-unes des causes que nous venons d'examiner, notamment à l'impaludisme, à la syphilis, à la scrofule et aux irritations répétées de la peau, il faut bien reconnaître que cette influence est restreinte. C'est la filaire qui nous donne la raison de la fréquence de l'éléphantiasis dans les pays chauds. Mais quelle qu'étendue que soit son action elle n'est pas exclusive, comme le veut Manson. L'existence de la maladie dans les climats tempérés. chez des indvidus qui n'ont jamais voyagé dans les contrées tropicales, suffit à le prouver (Besnier, Arnozaud (1). Pour soutenir l'opinion contraire, il faut, à l'exemple de sir J. Fayrer (2), nier l'iddntité de l'éléphantiasis nostras et de l'état pathologique endémique dans les pays chauds. Or, d'après le témoignage des médecins de toutes les parties du monde, la maladie est la même partout. Les trois cas publiés en 1878 par M. Besnier,ceux de MM. J. Hutchinson (3), Bryant, Holmes, Alcock,C. Jakson, Crocker, Strange (4), ne diffèrent en rien des types les plus exotiques (Myers, T. Fox). M. Birkersteth rapporte un cas de lympho-scrotum des mieux caractérisés (5) apparu chez un individu qui n'avait jamais quitté le Royaume-Uni.

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(1) Arnozan. — Journ. de méd. de Bordeaux, n° 10, p. 77.
(2) Fayrer. — The Lancet I, 1879, p. 267 et 433.
(3) Hutchinson. Idem, II, 1876, p. 282.
(4) Cités par Myers. Cust. Gaz. méd. rep. n° 32.
(5) Bikersteth. — Th. Lancet II, 1871, p. 187.

CHAPITRE III

Discussion de la théorie parasitaire.

La théorie de la cause parasitaire de l'éléphantiasis des Arabes a rallié de nombreux partisans dans toutes les parties du monde, surtout au Brésil et en Angleterre. Elle a rencontré aussi quelques adversatres et non des moins illustres pour ne citer que M. le professeur Guès (1) en France et M. E. Fox (2) en Angleterre. Ces deux auteurs ont formulé contre elle un certain nombre d'objections qui méritent la discussion. Les voici :

1^{re} objection. — Tous les membres d'une famille boivent la même eau, sont dévorés par le même moustique, pourquoi ne sont-ils pas tous atteints de l'une ou l'autre des maladies attribuées à la filaire ? Celle-ci a été trouvée chez des individus en parfaite santé, elle ne ne possède donc pas d'influence nuisible.

Les partisans du parasitisme filarien n'ont jamais regardé l'éléphantiasis et les affections connexes comme des conséqusnces nécessaires de la présence de l'helminthe dans l'organisme, mais comme des accidents, à la vérité. très fréquents.

Tous les individus qui boivent de l'eau contaminée avalent le ver et l'hébergent, mais tous ne présentent pas ces accidents. Tous les chiens qui sont infestés par des filaires n'ont pas des troubles circulatoire, tous les trichinés ne sont pas voués fatalement aux désordres bien connus. Quand il s'agit de petits parasites, dit Fayrer, les lésions du territoire envahi sont dues à leur accroissement, à leur activité et à leur déplacement.

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(1) Guès. — Hématurie trop. in Arch. méd. nav., XXVII, 1879.
(2) E. Fox. — Thc Laucet, I, 1878, p. 463.

2° objection. — L'éléphantiasis peut exister sans filaire, ce qui est prouvé par les cas sporadiques des pays tempérés et par ceux des pays chauds dans lesquels des observateurs expérimentés avouent l'avoir cherchée en vain

L'existence de l'affection dans les contrées où elle n'est pas endémique prouve que des causes autres que le parasitisme peuvent produire des états pathologiques semblables, en d'autres termes, que l'éléphantiasis ne reconnait pas une étiologie unique ; mais ce fait n'enlève pas à la filaire son influence dans la majeure partie des cas tropicaux.

Que des observateurs des plus habiles n'aient pas toujours réussi à trouver le ver chez les malades des pays chauds cela ne démontre pas son absence dans tous les cas qui ont fait l'objet de ces examens négatifs. La filaire est très difficile à découvrir en raison de ses faibles dimensions, de sa grande mobilité, de son extrême transparence ; elle est inaccessible aux recherches pendant les heures de jour, elle peut être dans le système lymphatique et non dans le sang, et vice versà; enfin, elle est quelquefois temporairement absente de la circulation même la nuit. Manson rapporte un cas où il a dû examiner le sang quatre fois avant d'y découvrir le parasite. Cependant, il y a indubitablement des cas où celui-ci est absent d'une façon permaneute. Mauson s'en est assuré en examinant plusieurs fois par jour le sang d'individus en convalescence d'ablation du scrotum. Ces faits, dit-il, prouvent que le ver n'existe pas au moment où on l'a cherché, mais non qu'il n'a jamais existé ; certains malades l'avait présenté avant l'opération. Cette disparition définitive s'explique par l'une ou l'autre des des raisons suivantes :

a) Les vers adultes ont pu mourir après avoir endommagé les canaux lymphatiques. On trouve dans l'œsophage du chien des tumeurs filariennes et des cicatrices, les vers qui les ont produites étant morts ou ayant disparu. Il peut en être vraisembloblement de même chez l'homme ;

b) Les vers peuvent-être vivants, mais en kystés dans une situation telle qu'il leur est impossible de déverser leurs produits de conception dans la circulation ;

c.) Ils peuvent être dans leur situation habituelle, mais un seul sexe est représenté, ou bien les deux sont présents, mais ils ne sont pas réunis. Une femelle de *filaria immitis* a été trouvée dans l'œsophage d'un chien, le mâle était absent, et, *vice versâ*, celui-ci a été rencontré sans celle-là. Dans de telles conditions, ils ne pouvaient procréer.

Dans l'éléphantiasis des membres, qui évolue longtemps avant d'être vu par le médecin, il est probable que l'absence de l'embryon résulte de la mort du ver-parent.

3ᵉ objection. — Si l'éléphantiasis est réellement produite par la filaire, comment expliquer l'inflammation locale et les accès fébriles qui l'accompagnent ; pourquoi ces phénomènes sont-ils intermittents ?

Ils ne dépendent pas de la filaire, du moins dans une certaine mesure ; on les observe aussi dans les cas sporadiques des zônes tempérées, quoique avec une fréquence et une intensité moindres. La même intermittence a été signalée dans l'éléphantiasis nostras (Myers). Il n'est donc pas besoin de faire intervenir le parasite pour l'expliquer dans les cas tropicaux.

4ᵉ objection. — Si la chylurie et l'éléphantiasis reconnaissent la même cause, la filaire, pourquoi la première de ces affections atteint-elle les blancs et la seconde les indigènes ? Le parasite jouant le rôle mécanique d'une embolie ne devrait pas faire cette distinction de races.

Lewis nous a appris, bien avant que cette critique ne fut formulée, que les indigènes lorsqu'ils souffrent de la chylurie dissimulent soigneusement leur mal, ils en nient obstinément l'existence ; il faut pour arriver à le constater chez eux avoir recours au cathetérisme. D'autre part, M. Clarac nous dit qu'à la Martinique la chylurie est aussi commune chez le noir que chez le blanc. S'il en est autrement ailleurs, le fait ne manque pas d'explication. Qu'y a-t-il d'étonnant, en effet, à ce que, chez l'immigrant européen qui fait abus de boissons alcooliques, ce soit surtout l'appareil urinaire qui est frappé, et que, chez le noir qui marche nu-pieds et nu-jambes, ce soit le plus souvent les parties périphériques. Le parasite ne peut faire de distinction de races, mais, on le comprend, il obstrue plus facilement un département lymphatique déjà amoindri et y réveille les phlegmasiques antérieures.

6ᵉ objection. — Comment expliquer la prédilection marquée que montre la filaire pour les membres inférieures et le scrotum.

Waring et Day, dans des statistiques qui comprennent mille cas, ont montré, en effet, que cinq fois seulement pour cent la maladie se présente dans d'autres parties du corps. Ces faits, loin d'être contraires à la cause parasitaire, nous font voir que la filaire, comme beaucoup d'autres entozoaires, a un habitat de prédilection. L'anguillule intestinale se rencontre en très grande abondance dans le duodénum, elle est rare dans le jéjunum, ce n'est qu'exceptionnellement qu'on la rencontre dans l'iléon. Le ténia inerme se fixe à peu de distance du pylore, la filaria loa se loge ordinairement entre la conjonctive et le globe oculaire. Sur 497 cas de dracontiase, le ver de Médine occupait 442 fois le membre inférieur ; l'ascaride lombricoïde séjourne de préférence dans l'intestin grêle ; les filaires du chien vivent l'une dans le ventricule droit et l'artère pulmonaire, l'autre dans des kystes appendus aux parois de l'aorte et de l'œsophage.

6ᵉ objection. — La non-contagiosité de l'eléphantiasis infirme dans une certaine mesure la théorie parasitaire (M. Corre).

Nous savons que l'œuf ou l'embryon des eutozoaires doit abandonner l'habitat propre à l'individu adulte pour que le cycle génétique de l'espèce puisse s'accomplir. La filaire obéit à cette loi universelle. La contagion n'est donc pas possible par l'intermédiaire de ses embryons, encore moins par celui de ses œufs, qui sont des produits d'avortement. L'est-elle par l'animal adulte ? sans doute, à la rigueur. Mais quel concours de circonstances il faut admettre pour concevoir la possibilité de cette transmission ! En supposant qu'elle se produise, comment savoir qu'elle a eu lieu, puisque le contaminateur et le contaminé vivent dans les mêmes conditions ? Ensuite, celui ci n'est pas voué fatalement à la maladie.

7° objection. — Les filaires n'ont pas été trouvées dans l'éléphantiasis pur, c'est-à-dire, non compliqué de varices lymphatiques, de chylodermie et de Chylurie. Il n'est pas prouvé que l'éléphantiasis pur et le lympho-scrotum soient la même maladie.

Cette critique a été formulée en 1878, par MM. T. Fox et Farquhar ; aujourd'hui, elle n'est plus fondée. Quant à l'identité du lympho-scrotum, et de l'éléphantiasis ordinaire, nous avons déjà rappelé les arguments qui militent en sa faveur.

8° objection. — L'unité manque à l'étiologie animée, les spécimens de filaires rencontrés par les differents observateurs n'appartiennent pas à la même espèce. Le ver observé en Amérique est vivipare, tandis que, en Afrique, il est ovipare.

Cette objection n'atteint pas la théorie parasitaire, elle ne soulève qu'une question d'helminthologie. De ce qu'il y aurait plusieurs espèces de filaires au lieu d'une seule, que pourrait-on conclure ? Qu'il reste à déterminer quelle est celle qui produit la maladie, ou bien à rechercher si toutes concourent à sa genèse. Mais il ne serait pas légitime d'en déduire qu'aucune de ces espèces n'en est la cause.

Cependant, est-il exact de dire que les spécimens décrits par les différents médecins n'appartiennent pas au même ver ? Ce n'est pas l'opinion de ces médecins. Les filaires rencontrées au Brésil sont regardées comme identique à ceux des Indes par Silva Lima (1), qui, dans une visite au *Royal Victoria Hospital* de Netley (Southampton) a eu l'occasion de voir les préparations envoyées de Calcutta par Lewis. Crevaux considère celles qu'il a vues aux Antilles comme des embryons d'un seul et même helminthe. Les neufs embryons que Lewis a recueilli en 1872 dans le sang d'un hindou atteint de diarrhée. étaient absolument semblables à ceux qu'il avait trouvés quatre ans auparavant. Sir J. Fayrer établit l'identité entre le spécimen que Sousino lui a montré en 1876 au Caire, et ceux de Lewis. L'éminent helminthologiste anglais, Spencer Cobbold, à propos d'un exemplaire adulte que lui a envoyé Baucroft, cette même année, admet la parenté de ce spécimen avec la forme embryonnaire. Les jeunes que Lewy a découvert en 1877 dans un cas d'éléphantiasis nœvoi du scrotum sont, en tout, comparables à ceux que lui, Cobbold, a vus dans d'autres parties du corps. Nous avons, ajoute celui-ci, assez de renseignements pour établir la relation génésique entre l'embryon de la filaire de l'homme, la filaire gastrique du moustique et la forme adulte et sexnée découverte par Baucroft.

En un mot tous les spécimens décrits par Wucherer, Salisbury, Lewis, Crevaux, Sousino, Silva Lima, Baucroft, Mauson, etc., doivent être rapportés à une seule et même espéce. (Cobbold). L'œuf a été trouvé aux Etats-Unis par Salisbury, et, chez un individu originaire de la Havane, par Demarquay.

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(1) S. Lima. Cité par B. Roncière in Arch. nav. XXX, p. 118.

9ᵉ objection. — Si on a rencontré la filaire adulte dans des tumeurs variqueuses du scrotum jamais encore une autopsie complète n'est venue prouver la réalité des lésions soupçonnées.

Les médecins des pays chauds ont eu, en effet, rarement l'occasion de faire des autopsies d'individus filariés, ils se sont toujours heurté dans cette voie aux préjugés des populations. Mais en l'absence d'autopsies complètes, ils ont eu les tumeurs et les membres qu'ils ont amputés. Au moyen de ces pièces ils ont montré que la maladie débute par la dilatation et l'obstruction des vaisseaux lymphatiques, l'engorgement des ganglions, l'infiltration des tissus Bien plus, ils ont découvert la filaire adulte dans l'épaisseur même des tissus affectés et déterminé son habitat propre. (Voir anatomie pathologique) L'éléphantiasis est une affection essentiellement locale.

OBSERVATIONS

OBSERVATION I (Personnelle).

Elèphantiasis de la jambe et du poignet.

Madame X....., 42 ans, originaire de la Guadeloupe, habitant Haïti depuis l'âge de 6 ans. Sa situation de fortune lui a toujours permis de mener une vie confortable. Elle n'a jamais eu de fièvre avant le début de l'affection actuelle, mais de 20 à 30 ans, ses vrines devenaient fréquemment blanches. Sauf à cette époque, sa santé à toujours été bonne ; constitution forte. Dans le cours de sa premiére grossesse, à 24 ans, elle a été prise d'un violent accès de fièvre qui a duré deux jours. En même temps, sa jambe droite toute entière est devenue douloureuse, rouge et enflée. Son médecin a cru à un érysipèle. Pendant sa seconde grossesse, à 26 ans, elle a eu une nouvelle attaque de fièvre et d'inflammation locale. Depuis cette époque ; ces phénomènes se sont renouvelés deux fois ou trois fois par an et à l'occasion de chaque grossesse, laissant le membre chaque fois plus tuméfié.

A sa cinquième grossesse, à 36 ans, l'inflammation s'est montrée au poignet en même temps qu'à la jambe et depuis, à chaque apparition, elle affectait les deux membres.

Nous voyons cette dame en mai 1890. Il y a trois ans que les accès fébroles n'ont pas reparu. La jambe est volumineuse (20 centimètres de plus que la gauche), déformée. presque uniformément cylindrique du genou au cou-de-pied. La peau dans toute cette étendue est blanche, luisante, tellement tendue qu'on ne peut la pincer ; elle semble adhérer intimement à la face interne du tibio. Elle forme, à la partie intérieure de la jambe, un bourrelet qui s'étend d'une malléole à l'autre. en passant en avant de l'articulation tibio-sarsienne.

Le poignet présente le même aspect que le membre intérieur mais ici la peau a conservé sa mobilité et ne forme pas de bourrelet ; elle semble doublée d'une forte couche de tissu induré. La tuméfaction s'étend à toute la face dorsale du poignet.

A ces caractères, nous avons reconnu l'éléphantiasis des Arabes, diaguostic qui a été, du reste, confirmé par MM. les professeurs G. Sée et Hardy.

Nous avons cherché la filaire dans le sang de cette maladie sans jamais la trouver. Néanmoins, nous croyons que son affection était sous la dépendance de ce parasite. En effet, cette dame vivait dans un pays où la filaire existe, elle a présenté a plusieurs reprises de la Chylurie. trouble qui est très souvent lié à la présence de l'eutozoaire, enfin elle n'avait dans ses antécédents aucune des maladies qui sont regardées par certains auteurs comme des causes suffisantes de l'éléphantiasis.

OBSERVATION II (Personnelle).

Lympho-scrotum et éléphantiasis du poignet. — Filaires dans le sang.

M. B...., 28 ans, commerçant à Haïti ; constitution faible ; pas de maladies antérieures graves, pas de fièvres intermittentes.

En 1886, il fut pris, sans malaise préalable d'un violent frisson accompagné de vomissements. Bientôt, il ressentit une douleur très vive au scrotum et dans les cuisses. La fièvre ne tarda pas à s'allumer et persista jusqu'au lendemain. Après la chute de la fièvre, il constata qu'il avait dans l'âme gauche des glandes de la grosseur d'un œuf de pigeon. Au bout de deux mois il eut un nouvel accès de fièvre, et depuis il est resté sujet à ces attaques fébriles et inflammatoires. Le sulfate de quinine a été impuissant à les prévenir. Elles venaient irrégulièrement tous les deux, trois ou quatre mois ; cependant depuis un an elles n'ont pas reparu.

En 1888, le malade s'est aperçu que son poignet gauche était plus lourd et plus gros que le droit. Il afirme pourtant que cette partie n'est jamais devenue ni rouge ni douloureuse.

Nous le voyons en août 1891. Ses bourses ont le volume d'une grosse orange, elles sont fortement piquenté et présente à leur surface de nombreuses élevures bulleuses, semi-transparentes, ressemblant à des grains de tapioca mal cuit. La région a conservé sa mollesse ; en pinçant la peau on sent qu'elle est doublée d'une épaisse couche de tissu peu consistant. Le testicule gauche est volumineux ; mais l'épaisseur des téguments ne permet pas de constater s'il y a une hyorocèle concomitante. Le malade n'a pas remarqué l'excès de volume de ce testicule avant le début de ses accés fébriles. Ses gauglious inguinaux gauches sont gros et durs, mais mobiles.

Le poignet gauche mesure 6 centimètres de plus que le droit. La peau est tendue, écailleuse et ne peut-être pincée ; le doigt n'y laisse pas son empreinte.

Un médecin distingué de Haïti, M. Aubry, ancien directeur de l'école de médecine de Port-au-Prince, à trouvé des filaires, en 1886, dans le sang de ce malade. Lui-même les a vues et les a compare à de petits serpents. Nous avons été moins heureux dans nos recherches, quatre examens de sang et de lymphe faite avec toutes les précautions indiquées par Lewis et Manson ne nous ont fourni aucun embryon de filaire. Quelle est la raison de ce résultat négatif ? Notre inhabileté mise de côté, il est vraisemblable que le parasite avait déjà disparu quand nous l'avons cherché. En effet, le début éloigné de la période hypertrophique permet de penser que l'obstruction était complète depuis longtemps. Nous savons ce qui se passe en pareil cas.

Ces deux observations nous montrent encore, d'une part la combinaison de l'éléphantiasis avec la chylurie ; d'autre part, celle de l'éléphantiasis ordinaire avec le lympho-scrotum et peut être avec l'hyorocèle chyleuse. affection qui rentre aussi dans le groupe de l'helminthiase filarienne.

OBSERVATIONS DE LEWIS (résumées.)

Obs. I. — Eléphantiasis du scrotum et chylurie ; filaires dans un caillot sanguin trouvé dans l'urine.

Obs. II. — Eléph. du scrotum datant de neuf mois ; pas de chylurie ; une douzaine de fil. dans le liquide extrait par piqûre des varices lymphatiques.

Obs. III. — Eléph. du scrotum et des pieds, chylurie ; fil. dans le sang des doigts et des orteils.

OBSERVATIONS DE MANSON

Obs. I. — Siah, 45 ans, laboureur, atteint de lympho-scrotum. Pendant un séjour d'une semaine à l'hôpital, le sang de cet homme fut examiné quotidiennement, les filaires n'y ont jamais fait défaut, leur nombre variait de 4 à 5 dans six préparations de sang.

Obs. II. — Tso, âgé de 50 ans, éléph. du scrotum, fil. dans le sang dès le premier examen.

Obs. III. — Toon, âgé de 60 ans, coolie porteur de chaises. Entre 20 et 30 ans, il devint sujet aux accès de fièvre et d'inflammation des bourses, apparaissant à intervalles irréguliers d'un mois à un an. Ces attaques ont toujours reparu plus ou moins fréquemment depuis cette époque ; quelquefois cependant, le malade en a été exempt pendant un an ou deux. Cette année, la fièvre est moins intense qu'autrefois, mais revient à intervalles plus rapprochés (4 à 5 jours). Elle ressemble à la fièvre intermittente avec ses trois stades de frisson, de chaleur et de sueur, mais en diffère par les caractères suivants ; il n'y a pas de régularité dans son apparition, elle vient tantôt la nuit, tantôt le jour, et ne répond pas aux types réguliers de quotidienne, de tierce ou de quarte. Chaque attaque dure environ cinq jours, et est accompagnée d'un léger gouflement du scrotum. Le malade n'a jamais eu ni abcès du scrotum ni chylurie. Ses bourses sont considérablement augmentées de volume et présentent à leur surface des lymphatiques variqueux qui, ponctionnées, fournissent une lymphe blanchâtre. Le sang contient des filaires en abondance.

OBSERVATION DE SIEGFRIED

Eléphantiasis du scrotum, pas de varices lymphatiques, ni de chylurie, ni de diarrhée ; hypertrophie et induration des ganglions inguinaux ; embryons et enveloppes de filaire dans la lymphe ganglionnaire.

CONCLUSIONS PARTICULIÈRES

A. — L'éléphantiasis des Arabes est, plus souvent que l'état de santé, associé à la présence de la filaire du sang dans l'organisme.

B. — Ce parasite est communément trouvé dans les vaisseaux et les ganglions lymphatiques.

C. - Ces vaisseaux sont dilatés et variqueux en certains points, imperméables dans d'autres ; ces glandes sont tantôt variqueuses, tantôt indurées, presque toujours hypertrophiées.

D. — L'éléphantiasis débute par le système lymphatique, comme le démontrent la clinique et l'anatomie pathologique.

E. — La pathogénie, généralement reconnue à cette affection, même par les auteurs qui la rapportent à des causes autres que la filaire, est l'obstruction d'un département lymphatique.

F. — La filaire est un corps étranger libre dans la circulation, elle peut donc jouer ce rôle d'agent obstruant.

CONCLUSION GÉNÉRALE

Il existe dans les rapports de la filaire du sang avec l'éléphantiasis des Arabes une relation de cause à effet qui nous explique l'endémicité de cette maladie partout où l'entozoaire a été observé.

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Vu et permis d'imprimer :
Le Vice-Recteur de l'Académie de Paris,
GRÉARD.

Paris. — Imp. de la Faculté de Médecine. Henri Jouve, 15, rue Racine.

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